

**REMEDIAL PLANNING ACTIVITIES AT
SELECTED UNCONTROLLED HAZARDOUS
SUBSTANCES DISPOSAL SITES IN A ZONE
FOR EPA REGIONS VI, VII, & VIII**

CONTRACT NO. 68-W9-0021



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CDM FEDERAL PROGRAMS CORPORATION
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SELECTED UNCONTROLLED HAZARDOUS
SUBSTANCES DISPOSAL SITES IN A ZONE
FOR EPA REGIONS VI, VII, & VIII

U.S. EPA CONTRACT NO. 68-W9-0021

FINAL

BASELINE HUMAN HEALTH RISK ASSESSMENT

ANACONDA SMELTER NPL SITE
ANACONDA, MONTANA

January 24, 1996

Work Assignment No: 037-8P18
Document Control No.: 7760-037-RA-DMCG

Prepared for:

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
Region VIII, Montana Office
Federal Building, Drawer 10096
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Prepared by:

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CDM FEDERAL PROGRAMS CORPORATION
a subsidiary of Camp Dresser & McKee Inc.

January 24, 1996

Mr. Charlie Coleman
USEPA, Region VIII, Montana Office
Federal Building, Drawer 10096
301 South Park Avenue
Helena, MT 59626

Project: ARCS Regions VI, VII, and VIII, Contract No. 68-W9-0021

DCN: 7760-037-RA-DMCG

Subject: Final Baseline Human Health Risk Assessment for the Anaconda
Smelter NPL Site

Dear Charlie:

Enclosed are 10 copies of the subject document for distribution. In addition, copies of the document have been sent to the following individuals.

Bob Alexander, CDM Federal
Bill Brattin, Weston
Julie DalSoglio, EPA
Susan Griffin, EPA
Jim LaVelle, CDM Inc.
Andy Lensink, EPA
Susan Walker, Ageiss
Andy Young, MDEQ

Please don't hesitate to contact me if you have any questions regarding this document.

Sincerely,

CDM FEDERAL PROGRAMS CORPORATION

Angela Fisher

Angela Fisher
Risk Assessor

Enclosures

cc: PF

LR ANAC 006/DMCG.LTR


CDM FEDERAL PROGRAMS CORPORATION
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January 24, 1996

Grant hours

Mr. Charlie Coleman
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Rosemary

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CDM FEDERAL PROGRAMS CORPORATION

Angela Fisher

Angela Fisher
Risk Assessor

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LR ANAC 006/DMCG LTR

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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

REGION VIII, MONTANA OFFICE
FEDERAL BUILDING, 301 S. PARK, DRAWER 10096
HELENA, MONTANA 59626-0096

Ref: 8MO

February 12, 1996

MEMORANDUM

SUBJECT: Final Baseline Human Health Risk Assessment
for the Anaconda Smelter NPL Site

FROM: Charles Coleman, RPM, 8MO
Susan Griffin, Toxicologist, 8HWM-SM

TO: Distribution

Enclosed, for public distribution, is the Final Baseline Human Health Risk Assessment for the Anaconda Smelter NPL Site. Risk assessments were previously performed by EPA for the Old Works/East Anaconda Development Area, Flue Dust and Mill Creek Operable Units. This document completes the risk assessment process for the Anaconda Smelter NPL Site by addressing risks in areas of the site not previously addressed under operable unit-specific assessments.

This risk assessment was prepared by CDM Federal Programs Corporation for EPA to evaluate the potential for adverse human health effects to occur as a result of exposure to chemicals from ongoing and historic releases from the Anaconda Smelter site. This assessment, utilizes to the extent practicable, data collected by the University of Cincinnati in their Arsenic Exposure Study conducted in Anaconda over the past several years. This risk assessment will be used in the sites last two remaining projects: Community Soils; and Anaconda Regional Water, Waste & Soils.

For further information regarding the risk assessment or the use of this information in the Superfund process at the Anaconda site, please contact Charles Coleman at (406) 441-1150 xt 261.

Enclosure

cc: Andy Lensink, 8ENF-L
Julie DalSoglio, 8MO
Susan Griffin, 8HWM-SM
Andy Young, MDEQ-ERD

Ref: 8MO

February 12, 1996

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Julie DalSoglio, 8MO
Susan Griffin, 8HWM-SM
Andy Young, MDEQ-ERD

FCD: February 12, 1996:cbc:charlie:hhra.dis

8MO
Coleman
2/12/96



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

REGION VIII, MONTANA OFFICE
FEDERAL BUILDING, 301 S. PARK, DRAWER 10096
HELENA, MONTANA 59626-0096

Ref: 8MO

April 8, 1996

Mr. William D. Crowley, Ph.D.
3615 Nelson Road
Bozeman, MT 59715

Dear Dr. Crowley:

As requested, enclosed is the Final Baseline Human Health Risk Assessment for the Anaconda Smelter NPL Site, dated January 24, 1996. If you have any questions, please feel free to call me at (406) 441-1150 xt 261.

Sincerely,

A handwritten signature in black ink, appearing to read "Charles Coleman", is written over the typed name.

Charles Coleman
Anaconda Project Manager

Enclosure

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SELECTED UNCONTROLLED HAZARDOUS
SUBSTANCES DISPOSAL SITES IN A ZONE
FOR EPA REGIONS VI, VII, & VIII

U.S. EPA CONTRACT NO. 68-W9-0021

FINAL

BASELINE HUMAN HEALTH RISK ASSESSMENT

ANACONDA SMELTER NPL SITE
ANACONDA, MONTANA

January 24, 1996

Prepared by:	<u>Angela Fisher</u>	<u>1/24/96</u>
	Angela Fisher Risk Assessor	Date
Prepared by:	<u>Jim LaVelle</u>	<u>1/24/96</u>
	Jim LaVelle Senior Risk Assessor	Date
Prepared by:	<u>Brigitte Howe</u>	<u>1/24/96</u>
	Brigitte Howe Risk Assessor	Date
Approved by:	<u>Robert R. Alexander AF</u>	<u>1/24/96</u>
	Robert R. Alexander Work Assignment Manager	Date

TABLE OF CONTENTS

<u>SECTION</u>	<u>PAGE</u>
LIST OF FIGURES	vii
LIST OF TABLES	vii
ABBREVIATIONS, ACRONYMS, AND INITIALISMS	ix
EXECUTIVE SUMMARY	ES-1
1.0 INTRODUCTION	1-1
1.1 OVERVIEW OF RISK ASSESSMENT	1-1
1.2 SITE BACKGROUND INFORMATION	1-2
1.3 SCOPE OF THE BASELINE HUMAN HEALTH RISK ASSESSMENT	1-7
2.0 IDENTIFICATION OF CHEMICALS OF POTENTIAL CONCERN	2-1
2.1 SITE-SPECIFIC DATA CONSIDERATIONS	2-1
2.1.1 AVAILABLE DATA	2-2
2.1.2 SELECTION OF DATA FOR USE IN THIS RISK ASSESSMENT	2-2
2.1.2.1 PTI (1992 and 1993)	2-7
2.1.2.2 Bornschein (1992 and 1994)	2-11
2.1.2.3 CDM Federal (1994a)	2-17
2.2 COMPARABILITY OF AVAILABLE DATA	2-18
2.3 BACKGROUND SAMPLING	2-19
2.3.1 BACKGROUND SOIL CONCENTRATIONS	2-20
2.3.2 BACKGROUND GROUNDWATER CONCENTRATIONS	2-21
2.4 SUMMARY OF DATA USED FOR QUANTITATIVE RISK ASSESSMENT	2-21
2.4.1 SUMMARY OF DATA ADEQUACY	2-21
2.4.2 SOILS	2-22
2.4.3 GROUNDWATER	2-22
2.4.4 DUST	2-23
3.0 EXPOSURE ASSESSMENT	3-1
3.1 CHARACTERIZATION OF SITE SETTING	3-2
3.1.1 PHYSICAL SETTING	3-2
3.1.1.1 Meteorology	3-2
3.1.1.2 Geology	3-3
3.1.1.3 Vegetation	3-4
3.1.1.4 Hydrogeology	3-4

TABLE OF CONTENTS (continued)

<u>SECTION</u>	<u>PAGE</u>
3.1.1.5 Hydrology	3-5
3.1.2 POTENTIALLY EXPOSED POPULATIONS	3-6
3.1.2.1 Current Site Conditions	3-6
3.1.2.2 Future Site Conditions	3-10
3.1.2.3 Subpopulations of Concern	3-11
3.1.2.4 Summary	3-11
3.2 IDENTIFICATION OF EXPOSURE PATHWAYS	3-12
3.3 QUANTIFICATION OF EXPOSURE	3-20
3.3.1 ESTIMATION OF EXPOSURE POINT CONCENTRATIONS ..	3-21
3.3.1.1 Soils	3-21
3.3.1.2 Dust	3-24
3.3.1.3 Groundwater	3-26
3.3.1.4 Data Manipulation	3-28
3.3.2 EXPOSURE ASSUMPTIONS	3-30
3.3.2.1 Exposure Assumptions Common to All Pathways	3-30
3.3.2.2 Site-Specific Exposure Assumptions	3-36
3.3.2.3 Standard Default Exposure Assumptions	3-38
3.3.3 CALCULATION OF CHEMICAL INTAKES — RESIDENTIAL RECEPTOR	3-41
3.3.3.1 Ingestion of Chemicals in Surface Soil and Interior Dust	3-42
3.3.3.2 Ingestion of Water	3-43
3.4 COMPARISON OF PREDICTED AND MEASURED EXPOSURES TO ARSENIC	3-45
3.5 UNCERTAINTIES ASSOCIATED WITH EXPOSURE ASSESSMENT ..	3-49
4.0 TOXICITY ASSESSMENT	4-1
4.1 TOXICITY CRITERIA	4-3
4.1.1 CARCINOGENS	4-3
4.1.2 NONCARCINOGENS	4-6
4.2 UNCERTAINTIES ASSOCIATED WITH TOXICITY ASSESSMENT ...	4-7
4.3 TOXICITY PROFILES	4-9
4.3.1 ARSENIC	4-9
4.3.2 LEAD (INORGANIC)	4-16
5.0 RISK CHARACTERIZATION	5-1
5.1 ARSENIC HEALTH RISKS	5-1
5.1.1 CANCER HEALTH RISKS FROM ARSENIC EXPOSURE	5-1

TABLE OF CONTENTS (continued)

<u>SECTION</u>	<u>PAGE</u>
5.1.2 NONCARCINOGENIC HEALTH RISKS FROM ARSENIC EXPOSURE	5-3
5.1.3 COMBINED RISKS	5-6
5.2 POTENTIAL HEALTH RISKS ASSOCIATED WITH EXPOSURE TO LEAD	5-6
5.3 ANALYSIS OF UNCERTAINTIES	5-20
5.3.1 LACK OF DATA FOR CHEMICALS OTHER THAN ARSENIC IN ANACONDA GROUNDWATER	5-21
5.3.2 LIMITED ENVIRONMENTAL DATA FOR AREAS OUTSIDE ANACONDA AND OPPORTUNITY	5-22
5.3.3 LACK OF DATA FOR LEAD IN INTERIOR DUST	5-22
5.3.4 UNCERTAINTY IN TOXICITY CRITERIA FOR ARSENIC ...	5-24
5.3.5 LACK OF DATA ON BIOAVAILABILITY OF LEAD IN SOILS AND INTERIOR DUST	5-28
5.3.6 LACK OF METHODOLOGY FOR EVALUATING DERMAL EXPOSURE TO METALS IN SOIL	5-29
5.3.7 USE OF DEFAULT EXPOSURE ASSUMPTION AND PROFESSIONAL JUDGEMENT	5-29
5.4 SUMMARY	5-30
6.0 RISK-BASED SCREENING LEVELS	6-1
6.1 EXPOSURE ASSUMPTIONS	6-2
6.1.1 RESIDENTIAL EXPOSURE SCENARIO	6-2
6.1.2 AGRICULTURAL WORKER SCENARIO	6-6
6.1.3 COMMERCIAL WORKER SCENARIO	6-13
6.1.4 RECREATIONAL VISITOR SCENARIO (ADOLESCENT PLAYING IN POOLED WATER)	6-17
6.1.5 RECREATIONAL VISITOR (DIRT BIKER) SCENARIO	6-20
6.2 SCREENING LEVELS FOR THE ANACONDA SMELTER NPL SITE ..	6-25
7.0 SUMMARY AND CONCLUSIONS	7-1
7.1 IDENTIFICATION OF CHEMICALS OF POTENTIAL CONCERN	7-1
7.2 EXPOSURE ASSESSMENT	7-1
7.3 HUMAN RISK CHARACTERIZATION	7-4
7.3.1 SUMMARY OF CANCER RISKS	7-5
7.3.2 SUMMARY OF NONCARCINOGENIC RISKS	7-5
7.3.3 LEAD TOXICITY	7-5
7.4 UNCERTAINTIES	7-5

TABLE OF CONTENTS (continued)

<u>SECTION</u>	<u>PAGE</u>
8.0 REFERENCES	8-1
8.1 GENERAL REFERENCES FOR THE RISK ASSESSMENT	8-1
8.2 REFERENCES FOR THE ARSENIC TOXICOLOGICAL PROFILE	8-6
8.3 REFERENCES FOR THE LEAD TOXICOLOGICAL PROFILE	8-9
APPENDIX A DATA EVALUATION	
APPENDIX B STATISTICAL COMPARISON OF PTI AND BORNSCHEIN SOIL DATA	
APPENDIX C DEVELOPMENT OF ARSENIC BIOAVAILABILITY FACTORS	
APPENDIX D COMPARISON OF PREDICTED AND MEASURED URINARY ARSENIC	
APPENDIX E LEAD MODELING RUN	

LIST OF FIGURES

<u>FIGURE</u>	<u>PAGE</u>
1-1 Anaconda Smelter NPL Site Map	1-3
2-1 Modified Bornschein Sub-Areas	2-13
3-1 Land Use in Anaconda	3-8
3-2 Land Use in Opportunity	3-9
3-3 Site Conceptual Exposure Model	3-13
3-4 Measured and Predicted Speciated Urinary Arsenic	3-48
5-1 IEUBK Modeling Blood Lead Results - Subarea A	5-8
5-2 IEUBK Modeling Blood Lead Results - Subarea B	5-9
5-3 IEUBK Modeling Blood Lead Results - Subarea C	5-10
5-4 IEUBK Modeling Blood Lead Results - Subarea D	5-11
5-5 IEUBK Modeling Blood Lead Results - Subarea E	5-12
5-6 IEUBK Modeling Blood Lead Results - Subarea F ₁	5-13
5-7 IEUBK Modeling Blood Lead Results - Subarea F ₂	5-14
5-8 IEUBK Modeling Blood Lead Results - Subarea I	5-15
5-9 IEUBK Modeling Blood Lead Results - Subarea J	5-16
5-10 IEUBK Modeling Blood Lead Results - Subarea G (Opportunity)	5-17
5-11 IEUBK Modeling Blood Lead Results - All Subareas Combined	5-18

LIST OF TABLES

<u>TABLE</u>	<u>PAGE</u>
ES-1 Risk-Based Screening Levels for Arsenic for the Anaconda Smelter Site	ES-7
ES-2 Noncancer Risks, Ingestion of Arsenic in Groundwater, Soil, and Dust, RME and CTE Residential Scenario	ES-8
ES-3 Cancer Risks, Ingestion of Arsenic in Groundwater, Soil, and Dust, RME and CTE Residential Scenario	ES-9
2-1 Summary of Main Data Sources	2-4
2-2 Summary of Statistical Data for Chemicals in Soil Comparison to Residential Soil Screening Criterion	2-5
2-3 Summary of Statistical Data for Chemicals in Groundwater Comparison to Tapwater Screening Criterion	2-6
2-4 Subarea Letter Descriptions Used in the Bornschein Study	2-12

LIST OF TABLES (continued)

<u>TABLE</u>	<u>PAGE</u>
3-1 Anaconda Summary Statistics for Soil Data	3-22
3-2 Summary of Soil Lead Data	3-23
3-3 Anaconda Summary Statistics for Dust Data	3-25
3-4 Calculated Concentrations of Lead in Indoor Dust	3-27
3-5 Arsenic Exposure Point Concentrations in Groundwater	3-29
3-6 Variables Common to All Exposure Equations (CTE)	3-31
3-7 Variables Common to All Exposure Equations (RME)	3-32
3-8 Variables Associated with Specific Pathways (CTE)	3-33
3-9 Variables Associated with Specific Pathways (RME)	3-34
3-10 Default Model Parameters	3-40
3-11 Chronic Daily Intakes, Ingestion of Arsenic in Groundwater, Soil, and Dust, RME and CTE Residential Scenario	3-44
4-1 Toxicity Values: Potential Carcinogenic Effects	4-5
4-2 Toxicity Values: Potential Noncarcinogenic Effects	4-8
5-1 Cancer Risks, Ingestion of Arsenic in Groundwater, Soil, and Dust, RME and CTE Residential Scenario	5-2
5-2 Noncancer Risks, Ingestion of Arsenic in Groundwater, Soil, and Dust, RME and CTE Residential Scenario	5-5
5-3 IEUBK Modeling Results Summary	5-19
6-1 Exposure Parameters for the Residential Scenario	6-3
6-2 Exposure Parameters for the Agricultural Worker Scenario	6-7
6-3 Exposure Assumptions for the Commercial Worker Scenario	6-15
6-4 Exposure Parameters for the Recreational Visitor Scenario	6-18
6-5 Exposure Assumptions for the Dirt Biker Scenario	6-21
6-6 Risk-Based Screening Levels for Arsenic for the Anaconda Smelter Site	6-26

ABBREVIATIONS, ACRONYMS, AND INITIALISMS

AA	atomic absorption
ADLC	Anaconda-Deer Lodge County
AIC	acceptable intake for chronic exposure
AOC	Administrative Order on Consent
ARCO	Atlantic Richfield Company
As	arsenic
AT	averaging time (days)
ATSDR	Agency for Toxic Substances and Disease Registry
AUC	area under the curve
AWQC	Ambient Water Quality Criteria
BAF	bioavailability factor for COPC in soil or dust (unitless)
BW	body weight (kg)
CDI	Chronic Daily Intake (mg/kg-day)
CDM Federal	CDM Federal Programs Corporation
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act of 1980
CF	conversion factor (10^{-6} kg/mg)
CFR	Code of Federal Regulations
CLP	Contract Laboratory Program
cm	centimeter(s)
COPC	chemical of potential concern
CRAVE	Cancer Review and Validation Effort (EPA)
CS	chemical concentration in soil or dust (mg/kg)
CSF	cancer slope factor
CTE	central tendency exposure
DMA	dimethyl arsenic acid
DPS	Development Permit System

ABBREVIATIONS, ACRONYMS, AND INITIALISMS (continued)

ED	exposure duration (years)
EE/CA	Engineering Evaluation/Cost Analysis
EF	exposure frequency (days/year)
EPA	U.S. Environmental Protection Agency
°F	degrees Fahrenheit
FI	fraction ingested from contaminated source (unitless)
HEAST	Health Effects Assessment Summary Tables (EPA)
HHRA	human health risk assessment
HI	Hazard Index
HQ	Hazard Quotient
ICP-MS	inductively coupled plasma-mass spectroscopy
IEUBK	Integrated Exposure Uptake Biokinetic
IQ	intelligence quotient
IR	ingestion rate (mg/day)
IRIS	Integrated Risk Information System
L/day	liters per day
LOAEL	lowest-observable-adverse-effect level
LOEL	lowest-observed-effect-level
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
mg/day	milligrams per day
mg/kg	milligram(s) per kilogram
mg/kg-day	milligram(s) per kilogram per day
mg/L	milligram(s) per liter
MMA	monomethyl arsenic acid
mph	miles per hour
NCDC	National Climatic Data Center
NCP	National Contingency Plan

ABBREVIATIONS, ACRONYMS, AND INITIALISMS (continued)

NCV	nerve conduction velocities
NHANES II	National Health and Nutrition Examination Survey
NOAEL	no-observable-adverse-effect level
NPL	National Priorities List
OU	Operable Unit
OW/EADA	Old Works/East Anaconda Development Area (operable unit)
Pb	lead
pH	negative log of the hydrogen ion concentration
ppm	parts per million
PRP	potentially responsible party
QA/QC	quality assurance/quality control
RfC	reference concentration
RfD	reference dose
RI/FS	Remedial Investigation/Feasibility Study
RME	reasonable maximum exposure
ROD	Record of Decision
RPM	regional project manager (EPA)
SCEM	site conceptual exposure model
SQL	sample quantitative limit
SST	Streamside Tailings
TCRA	time-critical removal action
UCL	upper confidence limit
XRF	X-ray fluorescence
%	percent
µg/L	microgram(s) per liter

EXECUTIVE SUMMARY

CDM Federal Programs Corporation (CDM Federal) has been tasked by the U.S. Environmental Protection Agency (EPA), Region VIII to evaluate the potential for adverse human health effects to occur as the result of exposure to chemicals from ongoing and historic releases from the Anaconda Smelter National Priorities List (NPL) Site.

Risk assessments were previously performed by EPA for the Old Works/East Anaconda Development Area (OW/EADA) Operable Unit (OU), the Flue Dust OU, and the Mill Creek OU. EPA also prepared an endangerment assessment, in which the health effects associated with exposure to chemicals transported by air from the Anaconda Smelter NPL Site were evaluated. The Atlantic Richfield Company (ARCO), the primary potentially responsible party (PRP), has contributed to the characterization of environmental contamination and human exposure at the Anaconda Smelter NPL Site. Their efforts include environmental sampling throughout the site and site-specific studies of urinary arsenic, soil ingestion, and arsenic bioavailability. This human health risk assessment (HHRA) completes the risk assessment process for the Anaconda Smelter NPL Site by addressing risks in areas of the site that were not addressed under an OU-specific risk assessment.

CHEMICALS OF POTENTIAL CONCERN

Chemicals of potential concern (COPCs) have been identified for OUs of the Anaconda Smelter NPL Site, and for other sites in the upper Clark Fork Basin. Arsenic, cadmium, and lead are ubiquitous in the area and elevated concentrations are generally associated with mining, milling, and smelting activities. In some instances, elevated concentrations of copper and zinc are also site-related and potentially hazardous. COPCs for the remaining areas of the Anaconda Smelter NPL Site are expected to be similar to those for OUs within the site and for sites in the region. Selection of COPCs is limited, therefore, to a determination of which of the above metals should be included in the quantitative assessment.

Data are available describing concentrations of arsenic, cadmium, copper, lead, and zinc in soil and groundwater. Data describing arsenic dust concentrations are also available. Soil concentrations of cadmium, copper, and zinc are below health-based screening levels; therefore, these chemicals will not be considered further in the risk assessment. Of the groundwater data available in areas where it is presently used for human consumption, only arsenic is present in concentrations indicating a potential health hazard. COPCs for the site are, therefore, arsenic and lead in soil and dust and arsenic in groundwater.

CHEMICAL CONCENTRATIONS IN SITE MEDIA

Data for this HHRA were provided by soil investigations conducted by PTI (1992 and 1993) and an arsenic exposure study conducted by Dr. Bornschein, University of Cincinnati (1992 and 1994). There were initial concerns that soil arsenic concentrations from the two studies were not comparable. Therefore, the data from both studies were used in a statistical comparison to determine if significant differences exist between arsenic concentrations found in surface soil samples collected for these two studies in Anaconda and nearby communities on an area-by-area basis. The Bornschein study divided the site into subareas, labeled A through K. Results of the statistical tests indicate that PTI (1992 and 1993) data and Bornschein (1992 and 1994) data are not significantly different for eight of nine areas compared. It is concluded that using either data set or a combination of the data will result in similar characterization of arsenic in surface soils for Anaconda and nearby communities. However, the Bornschein data are about eight times more numerous in Anaconda for subareas A through F than the PTI data, and samples were collected from yards where actual exposure to children may occur. The Bornschein data also included collocated interior dust samples. The PTI (1992 and 1993) soil samples were collected both from yards and pasture or idle land. Based on the above, EPA decided to use only the Bornschein data to develop exposure point concentrations of arsenic and lead in soil.

In addition, only the Bornschein study sampled water from the town of Anaconda. Bornschein (1992 and 1994) collected tapwater samples from homes using groundwater wells as their source of drinking water. These were located in subarea A. Samples were also collected from the public water supply. Tapwater samples were analyzed only for arsenic. The public water supply of the town of Anaconda is from an area considered uncontaminated and arsenic was not detected in samples collected from the public water supply. Therefore, for Anaconda subareas other than subarea A, which obtain drinking water from the public water supply, risks were calculated using an arsenic concentration of non-detect.

Groundwater investigations in the town of Opportunity were conducted by Bornschein (1992 and 1994) and CDM Federal (1994a). The water source for Opportunity is private domestic wells. The samples collected by Bornschein were analyzed only for arsenic. Samples collected by CDM Federal (1994a) were analyzed for metals and metalloids. Only arsenic was detected in concentrations presenting a potential health hazard. Due to the relatively small number of samples available, domestic groundwater data from both the CDM Federal (1994a) and Bornschein (1992 and 1994) studies will be used to develop exposure point concentrations for arsenic in groundwater in the town of Opportunity.

Bornschein (1992 and 1994) collected interior dust samples from the areas of Anaconda, Opportunity, Lost Creek, and Fairmont. These samples were analyzed for arsenic, which was present in concentrations indicating a potential health risk. Both arsenic and lead were retained as COPCs in soil; therefore, lead was also assumed to be a COPC in dust. Lead dust concentrations were estimated using a soil-to-dust ratio developed using arsenic soil-to-dust concentrations.

RECEPTORS AND EXPOSURE PATHWAYS

Based on current and future land uses, the following populations are considered most likely to be exposed to COPCs at the Anaconda Smelter NPL Site:

- Current and future residents
- Agricultural workers
- Recreational users
- Commercial workers

Exposure pathways of concern for these populations are:

Exposure Pathways for Residents (Adults and Children Ages 0 - 6):

- Ingestion of surface soils
- Ingestion of interior dust
- Ingestion of groundwater

Agricultural Workers (Adults):

- Ingestion of surface soils
- Inhalation of dust

Recreational Users (Dirt Bike Riders):

- Ingestion of surface soils
- Inhalation of dust

Recreational Visitors (Swimmers):

- Ingestion of surface water
- Dermal exposure to surface water

Commercial Workers (Adults):

- Ingestion of surface soils
- Ingestion of interior dust

COPC concentrations in soil and groundwater outside of the towns of Anaconda and Opportunity are not adequately characterized due to the relatively small number of samples collected. Therefore, only risks to current residents of Anaconda and Opportunity are assessed quantitatively in this HHRA. Risk-based screening levels are developed for other receptors to be used in risk characterization when data are available.

EXPOSURE ASSUMPTIONS

Arsenic chronic daily intake (CDI) was estimated for each residential exposure pathway based on estimates regarding the extent, frequency, and duration of exposures and the exposure point concentrations. Site-specific exposure assumptions were used when available; these include estimates of arsenic bioavailability in dust, soil, and water. EPA has used available data to derive site-specific arsenic bioavailability estimates for ingested soil and dust (EPA 1994a, 1995a). The following are the bioavailability values used in the HHRA:

- 25.8 percent (%) bioavailability for dust
- 18.3% bioavailability for soil
- 100% bioavailability for water

Findings in the Anaconda soil ingestion study support the Superfund Program's usual approach of assuming ingestion of 100 milligrams (mg) soil and dust per day as a central tendency exposure (CTE) assumption and 200 mg soil and dust per day as a reasonable maximum exposure (RME) assumption for ingestion rates of children 0-6 years old. Though default assumptions are used for soil and dust ingestion rates for children, these assumptions are clearly consistent with available site-specific data.

Predictions of exposure obtained from calculations of CDIs were compared to measured exposures of urine arsenic concentrations for children living in Anaconda. The arithmetic and geometric means of predicted and measured urinary arsenic concentrations for these children were compared to evaluate the appropriateness of the exposure assumptions used. The Kruskal-Wallis one-way analysis of variance demonstrated that measured and predicted urinary arsenic are not statistically different. However, EPA exposure calculations underpredict urinary arsenic concentrations where measured levels are greater than 10 micrograms per liter ($\mu\text{g/L}$). Overall, the results of the comparison support the use of the described exposure calculations in risk assessment for the Anaconda Smelter NPL Site.

RISK-BASED SCREENING LEVELS

Risk-based screening levels were developed for arsenic based on residential, agricultural and commercial worker and recreational swimmer and dirt biker exposure scenarios. Screening levels for the different exposure scenarios have been developed for a carcinogenic risk range of 10^{-7} to 10^{-3} and a noncarcinogenic hazard index (HI) of 1, and are provided in Table ES-1.

HUMAN HEALTH RISK CHARACTERIZATION

Toxicity values for arsenic were combined with CDI to estimate quantitative health risk estimates for exposure to arsenic. Lead toxicity was assessed using the EPA Integrated Exposure Uptake Biokinetic (IEUBK) Lead Model, Version 0.99. Children (aged 0-6) were considered the sensitive subpopulation at risk for adverse effects due to exposure to lead in environmental media. Risks to adults from lead exposure were not evaluated.

Noncarcinogenic risks were calculated by dividing the CDI of arsenic for each pathway by the arsenic-specific oral reference dose (RfD). The total noncancer risks for all pathways for each subarea is less than unity, indicating there is little potential for adverse noncarcinogenic effects (Table ES-2).

Carcinogenic risks were calculated by multiplying estimates of arsenic CDI by the arsenic-specific oral slope factor (SF). The total cancer risks for all pathways for each subarea fall within the range considered acceptable by EPA (Table ES-3).

Results of the IEUBK model indicate that 5% of children in subarea E may have blood-lead levels in excess of 10 micrograms per deciliter ($\mu\text{g/dL}$).

TABLE ES-1

Risk-Based Screening Levels for Arsenic for the Anaconda
Smelter Site

Medium	Soil								Surface Water	
Screening Level Based on Carcinogenic Risk	Residential Scenario (mg/kg)		Agricultural Scenario (mg/kg)		Commercial Worker Scenario (mg/kg)		Recreational Dirt Biker Scenario (mg/kg)		Recreational Youth/ Swimmer Scenario (mg/L)	
Carcinogenic Risk	RME	CTE	RME	CTE	RME	CTE	RME	CTE	RME	CTE
1×10^{-7}	0.30	1.85	1.00	10.04	1.33	10.15	2.32	53.55	0.002	0.008
1×10^{-6}	2.97	18.5	10.03	100.4	13.3	101.5	23.2	535.5	0.020	0.081
1×10^{-5}	29.7	185.2	100.3	1,003	133	1,015	232.3	5,355	0.20	0.81
1×10^{-4}	297	1,852	1,003	10,038	1,331	10,155	2,323	53,551	2.0	8.1
1×10^{-3}	2,970	18,516	10,033	100,385	13,307	101,546	23,231	535,517	20.2	81.0
Screening Level Based on Noncarcinogenic Effects (HI = 1)	573	1,071	NC	NC	2,139	4,570	NC	NC	1.04	4.16

NC = Not calculated. Risk-based screening levels for these exposure scenarios are based on inhalation and ingestion exposures. A RfC for inhalation is not available; screening levels based on noncarcinogenic effects can, therefore, not be calculated for these exposure scenarios.

TABLE ES-2
NONCANCER RISKS
INGESTION OF ARSENIC IN GROUNDWATER, SOIL, AND DUST
RME AND CTE RESIDENTIAL SCENARIO
ANACONDA SMELTER SITE
(mg/kg-day)

SUBAREA	RME SCENARIO	CTE SCENARIO
	Total Arsenic Risk	Total Arsenic Risk
Subarea A	5.48E-01	2.46E-01
Subarea B	2.79E-01	1.49E-01
Subarea C	3.60E-01	1.93E-01
Subarea D	5.70E-01	3.05E-01
Subarea E	3.80E-01	2.03E-01
Subarea F1	5.24E-01	2.80E-01
Subarea F2	4.48E-01	2.40E-01
Subarea I	3.45E-01	1.84E-01
Subarea J	3.32E-01	1.77E-01
Opportunity	6.03E-01	2.83E-01

TABLE ES-3
 CANCER RISKS
 INGESTION OF ARSENIC IN GROUNDWATER, SOIL, AND DUST
 RME AND CTE RESIDENTIAL SCENARIO
 ANACONDA SMELTER SITE
 (mg/kg-day)

SUBAREA	RME SCENARIO	CTE SCENARIO
	Total Arsenic Cancer Risk	Total Arsenic Cancer Risk
Subarea A	5.30E-05	6.38E-06
Subarea B	2.05E-05	3.23E-06
Subarea C	2.64E-05	4.17E-06
Subarea D	4.18E-05	6.59E-06
Subarea E	2.79E-05	4.40E-06
Subarea F1	3.84E-05	6.06E-06
Subarea F2	3.29E-05	5.19E-06
Subarea I	2.53E-05	3.98E-06
Subarea J	2.43E-05	3.83E-06
Opportunity	5.51E-05	7.01E-06

1.0 INTRODUCTION

1.1 OVERVIEW OF RISK ASSESSMENT

The U.S. Environmental Protection Agency (EPA) began initial investigations of the upper Clark Fork River Basin in 1982. These investigations prompted EPA to place the Anaconda Smelter on the National Priorities List (NPL) in 1983. Subsequent and ongoing remediation at this site has successfully addressed many of the waste sources, including flue dust, tailings, slag, and beryllium wastes associated with previous operations. However, chemicals released during smelter operations are still present in the environment near the former smelter. Fallout from past smelter emissions and transport of contaminants via wind and water, from existing tailings ponds, slag ponds, and other waste piles still present a potential risk to human health. EPA has initiated this baseline human health risk assessment (HHRA) to evaluate the potential for adverse effects to occur from exposure to chemicals from ongoing and historic releases to areas near the former smelter that have not been included in previous risk assessments.

Risk assessments were previously performed by EPA for the Old Works/East Anaconda Development Area (OW/EADA) Operable Unit (OU), the Flue Dust OU, and the Mill Creek OU. EPA also prepared an endangerment assessment in which the health effects associated with exposure to chemicals transported by air from the Anaconda Smelter NPL Site were evaluated.

Most data used to complete risk assessments for the Anaconda Smelter NPL Site have been gathered by the Atlantic Richfield Company (ARCO), which is the primary potentially responsible party (PRP). These data gathering efforts have contributed significantly to the characterization of environmental contamination and human exposure at the site. ARCO's efforts include environmental sampling throughout the site and site-specific studies of human arsenic exposure, soil ingestion, and arsenic bioavailability in experimental animals.

1.2 SITE BACKGROUND INFORMATION

The Anaconda Smelter NPL Site is located in southwestern Montana, at the southern end of Deer Lodge Valley, approximately 25 miles northwest of the city of Butte (Figure 1-1).

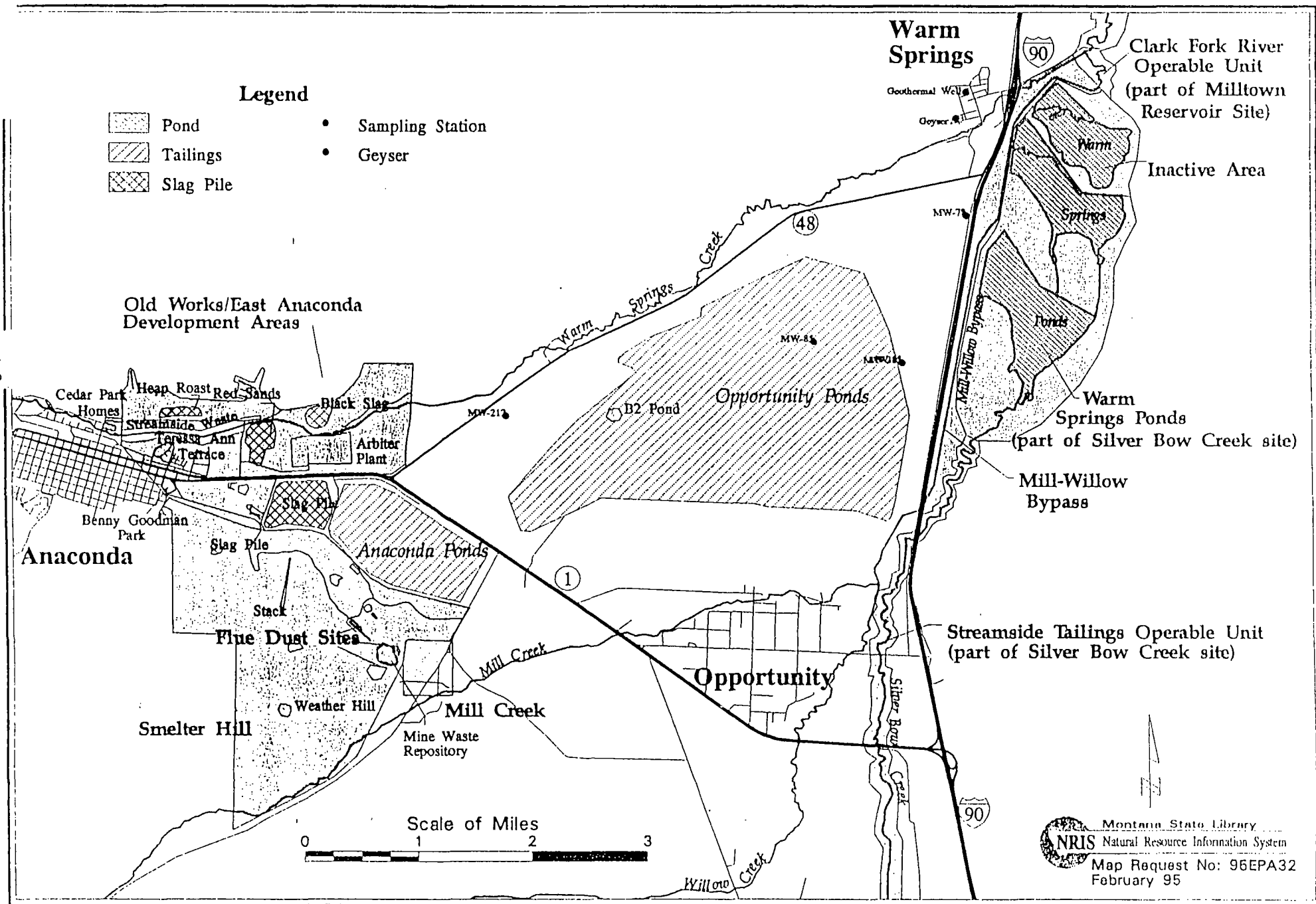
There are five communities located within the Anaconda Smelter NPL Site: Anaconda, Fairmont, Galen, Opportunity, and Warm Springs.

Around 1884, large copper concentrating and smelting operations commenced at the area presently known as the Old Works. The Old Works are located on the north side of Warm Springs Creek, east of Anaconda, and were operated until about 1901. In about 1902, ore processing and smelting operations began at the Anaconda Smelter (also called the Washoe Reduction Works, the Washoe Smelter, the New Works, and the Anaconda Reduction Works) on Smelter Hill south of Warm Springs Creek across from the Old Works. Operations at the Anaconda Smelter ceased in 1980, and the smelter facilities were dismantled soon thereafter. The only substantial feature remaining from the facility is the large brick smelter stack on Smelter Hill.

Activities at the Old Works and Anaconda Smelters and related facilities resulted in large volumes of waste materials, which were disposed of on the ground and in surface waters in and around the Anaconda Smelter NPL Site. Smelting activities also resulted in widespread, aerial deposition of contaminants released from stacks and from waste piles in the vicinity of the smelter, including the community of Anaconda. The history of significant releases of heavy metals to the environment at the Anaconda Smelter led to listing the site on the NPL in September 1983, under the authority of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA). On April 12, 1984, ARCO entered into an Administrative Order on Consent (AOC) with EPA to conduct demolition of the Smelter Hill facilities. In October 1984, ARCO entered into an AOC to conduct remedial investigations (RIs) for several OUs within the Anaconda Smelter NPL Site. Early draft

FIGURE 1-1

Anaconda Smelter NPL Site Map



1-3

reports based on initial investigations indicated wide-scale contamination and a need for more in-depth study.

In the initial stages of the Anaconda area investigations, it became apparent that the community of Mill Creek, located two miles east of Anaconda, was severely impacted by contamination. Children in Mill Creek had elevated urinary arsenic levels indicating an excess exposure to arsenic in their environment. EPA redirected the sequencing of the RIs for the site to focus on Mill Creek. Young children, the population determined to have the greatest exposure, were temporarily relocated from the community in May 1986. At this time, flue dust, the most concentrated arsenic and heavy metal contaminant source on the site, was sprayed with surfactant. In addition, contaminated road dust in the community was treated to reduce inhalation exposures. Following temporary relocation and the above remedial activities, none of the children from the Mill Creek area had levels of urinary arsenic above background.

In July 1986, EPA entered into an AOC with ARCO to conduct an expedited Remedial Investigation/Feasibility Study (RI/FS) for Mill Creek. The Record of Decision (ROD) for Mill Creek was completed by EPA in October 1987. The selected remedy was permanent relocation of Mill Creek residents. This remedy was chosen in part because of the potential for recontamination of the area from several nearby waste sources. EPA negotiated a Consent Decree with ARCO concerning the implementation of the relocation remedy for Mill Creek residents on January 7, 1988. The permanent relocation of residents was completed in the fall of 1988.

The generation and airborne transport of smokestack particulate and fugitive dust emissions during smelter operations also resulted in contamination of soils and household dust by arsenic, lead, cadmium, copper, and zinc in the neighborhoods of Teressa Ann Terrace, Elkhorn Apartments, Cedar Park Homes, and other areas surrounding the smelter. In

addition, contaminated material from the Old Works and/or Anaconda Smelter facilities was used as fill material around homes in the three Anaconda neighborhoods mentioned above.

On September 28, 1988, ARCO entered into an AOC with EPA to conduct an Engineering Evaluation/Cost Analysis (EE/CA) study and investigation for the Old Works and Community Soils OUs of the Anaconda Smelter NPL Site. Results of sampling conducted by ARCO in 1988-1989 in the areas of Teresa Ann Terrace, Elkhorn Apartments, and Cedar Park Homes indicated the presence of elevated heavy metal concentrations at or near the soil surface. Sampling conducted by ARCO in 1990 confirmed the presence of elevated concentrations of heavy metals in several yards, gardens, and common areas of the three neighborhoods.

In October 1988, EPA entered into an AOC with ARCO to conduct additional remedial and removal activities at the Anaconda Smelter NPL Site. Remedial investigations for the Flue Dust and Smelter Hill OUs were initiated at the same time as a removal analysis for the Old Works and Community Soils OUs. In March 1990, EPA and ARCO amended the October 1988 AOC to conduct an additional removal analysis at the Arbiter and Beryllium OUs. Further, EPA and ARCO agreed to conduct a siting analysis for a waste repository on the Anaconda Smelter NPL Site, and a site on Smelter Hill was eventually selected.

In 1990, ARCO entered into an AOC to conduct separate Old Works and Anaconda Community Soils investigations, and to conduct additional sampling in yards, gardens, and common areas of Teresa Ann Terrace, Elkhorn Apartments, and Cedar Park Homes neighborhoods. Sample analysis results confirmed that several yards and common areas contained elevated heavy metal concentrations at or near the soil surface. Arsenic concentrations ranged between 5 and 1,570 parts per million (ppm), cadmium ranged between 0.4 to 59.4 ppm, and lead ranged from 4.8 to 1,230 ppm.

A September 17, 1991, Enforcement/Action Memorandum (with a concurrent AOC) required ARCO to conduct a Time-Critical Removal Action (TCRA) by excavating and removing

contaminated soils in areas of Teresa Ann Terrace, Elkhorn Apartments, and Cedar Park Homes where arsenic concentrations exceeded 250 milligrams per kilogram (mg/kg). The proximity of these residential areas to the former Old Works smelting facilities may have contributed to the high levels of arsenic found. The primary objective of the action was mitigation of any direct contact threat to residents by removing the contaminated soil and replacing or capping the area with uncontaminated soil.

Under the TCRA, removal of arsenic-contaminated soils and replacement of topsoil and grass began in late 1991 and was completed in September 1992. The removed soils were disposed of in the Red Sands area of the OW/EADA OU. Removal occurred on about 8 acres of undeveloped lots and 19 front or back yards in Teresa Ann Terrace, 32 yards around the Elkhorn apartments, and 14 yards around Cedar Park Homes. Clean replacement soil was obtained from an area near Lost Creek.

From July through December 1992, material from two ponds east of the Arbiter Plant, four concrete bunkers behind the plant, and tailings from the Old Works Tailings Pond were excavated and taken to Smelter Hill for disposal in the Arbiter repository. Also during this time period, beryllium-contaminated tailings and other materials were removed and taken to a special repository constructed for these wastes.

In December 1992, EPA and ARCO entered into the Flue Dust Consent Decree, which resulted in the removal, stabilization, and placement in a repository of flue dust materials on Smelter Hill. This work was initiated in late 1992 and completed in 1994. In 1992, EPA and ARCO entered into an AOC to conduct the OW/EADA OU investigations. A March 1994 ROD selected a combination of engineering and institutional controls as the remedy. Remediation of recreational and commercial/industrial areas were conducted where waste and soils exceeded arsenic levels of 1,000 and 500 ppm, respectively. In early 1994, EPA began the scoping process for the final HHRA, culminating in the preparation of this report.

1.3 SCOPE OF THE BASELINE HUMAN HEALTH RISK ASSESSMENT

Risk assessments were previously performed by EPA for the OW/EADA OU, the Flue Dust OU, and the Mill Creek OU. This HHRA will complete the risk assessment process for the Anaconda Smelter NPL Site by addressing risks in areas of the site that were not addressed under an OU-specific risk assessment. The HHRA is based on information developed during the RIs, including the exposure and bioavailability studies sponsored by ARCO.

This HHRA quantitatively evaluates potential human health risks associated with site chemicals of potential concern (COPCs) found in soil, dust, and groundwater in areas of the Anaconda Smelter NPL Site not previously addressed in a risk assessment. The assessment is a baseline evaluation and assumes no further remedial action at the site. The no-action alternative is evaluated in accordance with Section 330.430(d) of the National Contingency Plan (NCP). The format and procedures used to develop this HHRA are based on the most recent EPA guidance for performing human health and evaluations at Superfund Sites (EPA 1989a, b; 1991a, b, c; 1992a, b, c; 1993a, b, c; 1994b, c).

A site conceptual exposure model (SCEM) for the Anaconda Smelter NPL Site is presented in this report. The SCEM includes potential current and future exposure pathways, and is presented in the form of an iterative flow chart, which depicts specific site characteristics including: (1) contaminant sources; (2) release mechanisms; (3) transport routes; (4) exposure routes; and (5) receptors. Exposure pathways identified in the SCEM for potential application to the site are individually evaluated for potential contribution to site-related exposure. Only those pathways that could present a significant risk to human health are evaluated quantitatively in this HHRA. For each such pathway, a complete description of receptors and exposure parameters are provided. Exposure parameters that deviate from defaults provided in EPA guidance documents are justified and referenced.

Toxicity profiles are provided for arsenic and lead, the only COPCs for this assessment. Each profile describes basic toxic properties and toxicokinetics and, where appropriate, provides EPA toxicity criteria. Toxicity criteria are not available for lead, since assessment of lead risks is now carried out using a physiologically-based, pharmacokinetic model.

Finally, carcinogenic and noncarcinogenic risks resulting from exposure to arsenic are estimated by combining estimates for arsenic exposure and toxicity criteria for arsenic. Risks from exposure to lead are estimated through use of the EPA Integrated Exposure Uptake Biokinetic (IEUBK) Lead Model Version 0.99. Uncertainties in all exposure and risk estimates are a critical part of risk characterization and are presented along with risk estimates to provide the risk manager with the appropriate perspective for applying risk information to remedial decisions.

2.0 IDENTIFICATION OF CHEMICALS OF POTENTIAL CONCERN

COPCs are chemicals that are potentially site-related and for which data are of sufficient quality for use in quantitative risk assessment (EPA 1989a). Selection of COPCs requires an evaluation of available data for useability in risk assessments, and a formal process for identifying site-related chemicals that might pose unacceptable risks at a site. COPCs have been previously identified for the Anaconda Smelter NPL Site and for other sites in the upper Clark Fork Basin. Arsenic, cadmium, and lead are ubiquitous in the area and elevated concentrations are generally associated with mining, milling, and smelting activities. In some instances, elevated concentrations of copper and zinc are also site-related and potentially hazardous. Based on this experience at other sites, the majority of sampling and analytical efforts at the Anaconda Smelter NPL Site focused specifically on these five chemicals, which are considered to be the primary COPCs at this site. Although some studies did collect data on other metals that might conceivably contribute to risk (e.g., antimony, barium, beryllium, manganese, and mercury), the relative contribution of these other chemicals to total risk is believed to be sufficiently small compared to the risks from the primary COPCs that they are not considered further. Selection of COPCs is limited, therefore, to a determination of which of the above metals should be included in the quantitative assessment.

2.1 SITE-SPECIFIC DATA CONSIDERATIONS

A large quantity of environmental data has been collected in the community of Anaconda and other areas addressed in this HHRA. In addition, site-specific studies of human exposure to arsenic and bioavailability of arsenic in experimental animals have been undertaken to support risk assessment at the site. In this section, available data are identified and evaluated for usefulness in quantitative risk assessment.

2.1.1 AVAILABLE DATA

Numerous investigations have been performed to provide data on the types and concentrations of chemicals in soil, dust, sediments, air, groundwater, surface water, and waste (tailings/slag) at the Anaconda Smelter NPL Site. Analyses have been performed using standard EPA Contract Laboratory Program (CLP) protocols as well as by X-ray fluorescence (XRF). Data were validated in accordance with procedures approved for the Clark Fork River Superfund Sites. Each data summary report prepared by ARCO was audited by EPA to determine the useability of each data point. EPA and ARCO have stipulated to the use of these data. As required for Clark Fork River Superfund Site investigations, data are classified into three data utilization categories: enforcement quality data, screening quality data, and unusable data. Enforcement quality data may be used for all Superfund program activities and purposes, screening quality data may be used for certain activities, and unusable (rejected) data are not usable for any Superfund purpose. Enforcement and screening data generated for Anaconda remedial investigations were used for the following purposes: site characterization, evaluation of alternatives, engineering design, risk assessment, determining presence or absence of contaminants, determining relative concentrations, and scoping and planning for future studies, investigations, or actions.

2.1.2 SELECTION OF DATA FOR USE IN THIS RISK ASSESSMENT

Prior risk assessments have been performed for the OW/EADA, Flue Dust, and Mill Creek OUs within the Anaconda Smelter NPL Site. This HHRA completes the risk assessment process by evaluating risks in areas of the Anaconda Smelter NPL Site that were not previously addressed. Data pertinent to these areas of the site are evaluated for useability in quantitative risk analysis. Data evaluation considers the following issues:

- Source and recentness of data
- Sampling locations
- Adequacy of documentation

- Data validation results
- Adequacy of analytical methods
- Detection limits
- Completeness
- Comparability

Reports containing data used for this HHRA include the Anaconda Soil Investigation (PTI 1992 and 1993), Anaconda Residential Urinary Arsenic Study (Bornschein 1992 and 1994), and CDM Federal (1994a). These studies are summarized in Table 2-1 and are evaluated in more detail below.

Data are available describing concentrations of arsenic, cadmium, copper, lead, and zinc in soil and groundwater. Table 2 - 2 provides a statistical summary of data for these chemicals in soil and compares concentrations to health-based screening criterion. As shown in Table 2 - 2, even maximum soil concentrations of cadmium, copper, and zinc are at or below health-based screening levels; therefore, these chemicals are not considered further in the risk assessment. The average concentration of lead is also less than the screening level; however, subareas within the town of Anaconda have soil lead concentrations higher than the screening level. Since subareas of the town have been identified as appropriate exposure units for this risk assessment (see Section 2.1.2.2), the higher average concentrations within these areas suggest a potential for unacceptable exposures for significant subpopulations within Anaconda. Based on mean lead concentrations within these areas, lead is retained as a COPC.

Table 2 - 3 provides summary statistics for groundwater data and compares concentrations to health-based screening criteria. Based on these comparisons, only arsenic is present in concentrations indicating a potential health hazard.

COPCs for the site are, therefore, arsenic and lead in soil and dust and arsenic in groundwater. Soil and groundwater data for these contaminants (Table 2 - 1) are evaluated below and used in Section 3.3.1 for estimating potential site-related exposures.

TABLE 2-1

Summary of Main Data Sources

Source	Description of Study
Anaconda Soil Investigation (PTI 1992a)	In order to characterize soil contamination in the Anaconda Smelter NPL Site, soil samples were collected from communities, including Anaconda, Opportunity, Warm Springs, Galen, and Fairmont. Areas of high use were targeted for additional sampling in Anaconda. Soil samples were also collected from areas near the communities, regional areas, and regional targeted areas such as streams, gulches, and the Yellow Ditch. Soil samples were analyzed for arsenic, cadmium, copper, lead, and zinc.
Anaconda Residential Urinary Arsenic Study (Bornschein 1992 and 1994)	To determine the extent of childhood arsenic exposure in residential environments in the Anaconda Smelter NPL Site, samples of soil, interior dust, exterior dust, and tapwater were collected from homes in Anaconda, Opportunity, Lost Creek, and Fairmont Ranches. Urine samples were collected from children living in these homes. Soil samples were analyzed for arsenic and lead; dust, water, and urine samples were analyzed for arsenic.
Domestic Water Sampling in and near Opportunity, Montana (CDM Federal 1994a)	A total of 20 groundwater samples were collected from 20 domestic wells in and near Opportunity. Samples were analyzed for metals and metalloids; lead data were not enforcement quality.

TABLE 2-2

Summary Statistical Data for Chemicals in Soil
Comparison to Residential Soil Screening Criterion¹

Chemical	Mean (mg/kg) (Sample No.)	Range of Concentrations Detected (mg/kg)	Screening Criterion for Residential Soil (mg/kg)
Arsenic	172 (318) ^a	38 - 409 ^a	0.37 ^b
Cadmium	5.01 (129) ^c	0.25 - 32.5 ^c	39 ^b
Copper	1337 (75) ^c	90 - 5070 ^c	9,990 ^d
Lead	384 (318) ^a	23 - 2153 ^a	400 ^b
Zinc	1662 (75) ^c	109 - 5210 ^c	23,000 ^b

^aSource: Bornschein (1992 and 1994)

^bSource: EPA 1994e

^cSource: PTI (1991 and 1992)

^dCalculated by CDM Federal

¹Soil screening criteria are based on RME scenarios. Soil ingestions is the only pathway evaluated. Dust is equivalent to soil in these calculations.

TABLE 2-3

**Summary Statistical Data for Chemicals in Groundwater
Comparison to Tapwater Screening Criterion¹**

Chemical	Range of Concentrations Detected (µg/L)	Screening Criterion for tapwater (µg/L)
Arsenic	1.0U - 13.8 ^a	0.038 ^b
Cadmium	0.1 - 0.8 ^c	18 ^b
Copper	1.9U - 10.8 ^c	1,400 ^b
Lead	None Detected ^c	15 ^d
Zinc	5.0U - 114.5 ^c	11,000 ^b

^aSource: CDM 1994a and Bornschein (1992 and 1994)

^bSource: EPA 1994e

^cSource: CDM 1994a (Bornschein data contained only As concs.)

^dAction Level

U = not detected

¹Screening criterion based on RME scenario. No exposure pathways other than groundwater ingestion are included in the calculations.

2.1.2.1 PTI (1992 and 1993)

The soil investigation carried out by PTI characterized concentrations and spatial distribution of arsenic and metals in several areas near the former Anaconda Smelter. These areas include: (1) communities of Anaconda, Opportunity, Warm Springs, Galen, and Fairmont; (2) community target areas in Anaconda; (3) locations near the communities of Anaconda, Opportunity, Warm Springs, Galen, and Fairmont; (4) regional areas; and (5) regional target areas.

Community Soils

Soil samples totaling 97 were collected at 76 sampling stations within the communities of Anaconda, Opportunity, Warm Springs, Galen, and Fairmont. Of the 76 sampling stations, 30 were located in the town of Anaconda, 28 were located in Opportunity, 12 were located in the town of Warm Springs, and 3 each were located in the towns of Galen and Fairmont.

Seventy-nine of the 97 samples were collected from the 0 - 2 inch interval and are considered surface soil samples. The remaining samples were collected from the 2 - 10 inch interval to determine the extent of vertical migration of contaminants. These deeper samples are considered subsurface soil. Samples were collected from yards, and included composite soils from lawn areas. In addition, samples were collected from pasture and idle land located within town limits. Arsenic and lead samples were analyzed by XRF, and cadmium by CLP methods. CLP methods were also used to determine soil moisture, pH, and electrical conductivity.

Community Target Areas

A total of 51 samples were collected from 24 sampling locations in the community of Anaconda. Community target areas were defined as areas having the potential for extensive

public use. These areas included schools, residential yards, public parks, and playgrounds. No community soils target areas were identified in the towns of Opportunity, Warm Springs, Galen, and Fairmont. Thirty-two of the 51 samples were composites collected from the 0 - 2 inch interval, 16 were discrete opportunistic samples from the 0 - 2 inch interval, and 3 were composite opportunistic samples from the 0 - 2 inch interval. All samples are considered surface soil. Analyses were performed as described above.

Near Community Soils

Near community sampling areas are defined as 0.5-mile strips surrounding the five individual communities defined by the community soils sampling effort. A total of 117 soil samples were collected at 94 sampling stations. Of these, 40 samples were collected near Anaconda, 33 samples were collected near Opportunity, 16 samples were collected near Warm Springs, and 12 samples each were collected near Galen and Fairmont. These soil samples were analyzed for arsenic, cadmium, and lead as described above. Soil slurry pH and electrical conductivity were measured in a field laboratory. Of the 117 samples collected, 113 were composite samples, and 4 were grab opportunistic samples. Ninety-three of the 113 samples were collected from the 0 - 2 inch interval and are considered surface soil. Eighteen of the remaining samples were collected from the 2 - 10 inch interval to determine the extent of vertical migration of contaminants and are considered subsurface soil samples. Two samples were collected from the 0 - 10 inch interval. The remaining four soil samples were opportunistic surface samples collected at two near community sample stations.

Regional Soils

The regional area consists of nearly 77,000 acres and includes undisturbed lands, such as native rangeland and riparian zones, and disturbed lands characterized as agricultural or reclaimed land. Composite soil samples were collected from a total of 84 stations on a 1 square mile grid corresponding to section corners designated by the General Land Office grid

system (PTI 1992). Seventy-four of the 84 sample stations were defined as undisturbed (rangeland) and 10 stations were defined as regional disturbed sites (tilled/reclaimed). Of these 10 stations, 5 were reclaimed waste sites and 5 were tilled agricultural lands. Arsenic, copper, lead and zinc were analyzed using XRF methods, and cadmium by CLP methods. Soil slurry pH and electrical conductivity were determined in a field laboratory.

A total of 100 samples was collected; 90 were considered to represent regional undisturbed areas and 10 were considered to represent regional disturbed areas. Seventy-one of the 90 regional "undisturbed" samples were collected from the 0 - 2 inch interval and are considered surface soil. Nineteen of the remaining samples were collected from the 2 - 10 inch to determine extent of vertical migration of contaminants and are considered subsurface soil. Regional "disturbed" samples were all collected from the 0 - 10 inch depth interval. These samples are considered subsurface soil.

Regional Targeted Soils

Soil samples were collected at a total of 60 sample station transects designated as regional targeted sampling locations. Regional targeted sampling areas are defined as perennial streams, ephemeral drainages, and other areas within the regional boundaries (e.g., Yellow Ditch) possibly contaminated by arsenic and metals. Sampling locations consisted of a total of 60 station transects across five creeks, seven gulches, and the Yellow Ditch. The entire reach of each stream or gulch was characterized using sample stations located at evenly spaced distances across the area. Soil samples totaling 246 were collected. Arsenic, copper, lead and zinc were analyzed by XRF methods, and cadmium by CLP methods. Soil slurry pH and electrical conductivity were determined in a field laboratory.

Data Evaluation

Following an evaluation of the PTI (1992 and 1993) data, EPA determined that samples were collected and analyzed, and data were validated and properly qualified, according to the procedures set forth in planning documents and EPA guidance.

The majority of samples were collected from surface soil (0 - 2 inches). Receptors in Anaconda and the surrounding areas are much more likely to come into contact with this soil than with deeper soils. Thus, the majority of soil data collected by PTI is directly applicable to exposures at the site.

Sample locations and numbers of samples within the towns of Anaconda and Opportunity are sufficient for defining exposure. The samples are reasonably closely spaced and it is unlikely that significant "hot spots" for arsenic and metals exist in unsampled areas. This conclusion is supported by an examination of the data collected by Bornschein (1992, 1994, see Section 2.1.2.2). Sample locations within other areas may not be representative because of the small number of samples collected. The areas in question are large and samples are widely spaced. It is conceivable that hot spots of significant size could be present in some of these areas. However, the examination of data from the communities of Anaconda and Opportunity suggests a rather homogeneous distribution of contamination. If the same mechanisms of contaminant release are responsible for contamination outside these communities, it is reasonable to expect a relatively homogeneous contaminant distribution in these areas also. Thus, the uncertainties in data for areas outside Anaconda and Opportunity may not be as great as suggested by simple examination of sample numbers and locations. Nevertheless, no quantitative evaluation of exposures is presented for communities or areas other than Anaconda and Opportunity. Additional discussion of potential exposures and risks in outlying and surrounding areas is presented in Section 6.0.

Summary

Data collected by PTI (1992 and 1993) are generally acceptable for use in this HHRA. Samples were collected and analyzed by acceptable methods, and data have been adequately validated. Only samples representing surface soil are considered appropriate for calculation of exposure point concentrations, however. Further, a sufficient number of samples were collected for risk characterization only for the communities of Anaconda and Opportunity. The number of samples collected from regional soils and other communities are considered insufficient to provide a quantitative risk assessment. However, since contaminant distributions are expected to be relatively homogeneous, the latter data may provide a useful general characterization of soil concentrations throughout the site.

2.1.2.2 Bornschein (1992 and 1994)

Bornschein (1992 and 1994) collected environmental samples, including soil, indoor dust, and tapwater in Anaconda, Opportunity, Lost Creek, and Fairmont. The Anaconda area was separated into several subareas for this investigation to ensure a more representative characterization of childhood exposure (Table 2-4). The other communities were retained as individual subareas. Within each subarea, samples were collected from single-family homes or multi-family buildings. For the purposes of this risk assessment, Subarea F was split into Subareas F₁ and F₂ (Figure 2-1).

Composite soil samples were collected to a depth of 2 centimeters (cm) from several different types of surface conditions within the yard of each home or building, including the perimeter of the home or building, garden areas, play areas, and bare areas of the yard. For large multi-family buildings, proportionately more composite samples were taken. Composite indoor dust samples were collected, using a small vacuum pump, from 3 areas within each home or apartment. These areas were intended to represent areas frequented by children, and included a floor area directly inside the main entry to the home, a floor area in the most frequently

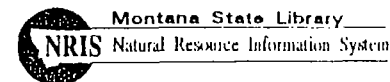
TABLE 2-4

Subarea Letter Descriptions Used in the Bornschein Study

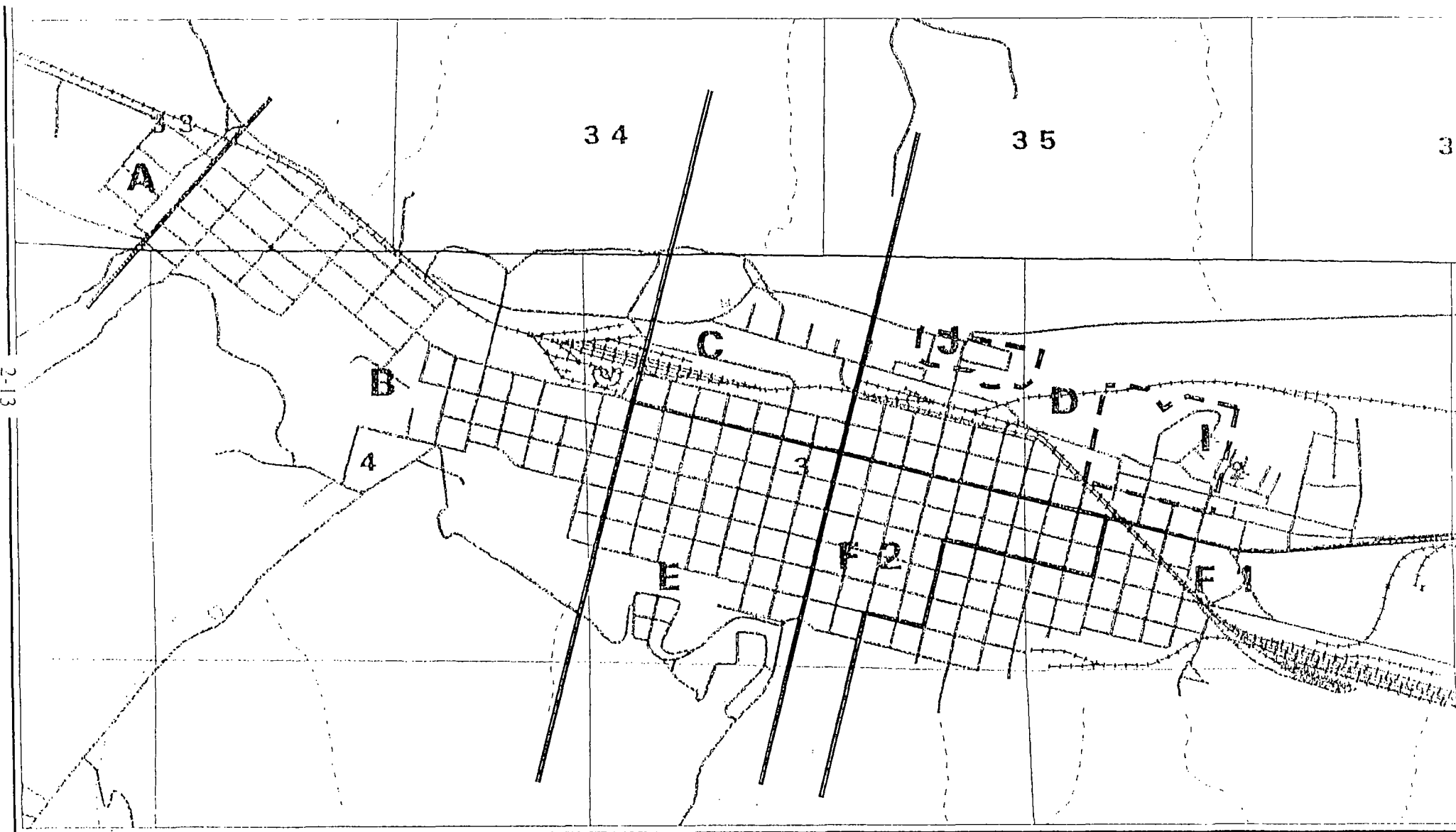
Subarea	Description
A	This area is bounded by the west side of Evergreen Street through the West Valley, including North Cable Road, English Gulch, and American Gulch.
B	This area is bounded by the east side of Evergreen Street through the west side of Spruce Street, including the Sunnyside Addition.
C	This area is bounded by the east side of Spruce Street, north side of Park Street, and west side of Main Street, including Mount Haggin Homes.
D	This area is bounded by the east side of Main Street, north side of Park Street, and the edge of town. Excluded from this area are Area I (Teressa Ann Terrace) and Area J (Cedar Park Homes). Areas I and J are special abatement areas.
E	This area is bounded by the east side of Spruce Street, south side of Park Street, and west side of Main Street.
F	This is bounded by the east side of Main Street, south side of Park Street, and edge of town.
G	This area is the community of Opportunity, bounded by the north side of Highway 1, including all of Opportunity.
H	This area is Lost Creek, including all homes on Galen Road.
I	This area is Teressa Ann Terrace. Teressa Ann Terrace is on the east end of town and includes all homes on Elaine Drive, Heather Drive, Jefferson Way, and Pauline Drive.
J	This area is Cedar Park Homes which includes all Cedar Park Homes low-income housing, homes on North Cedar Street, north of the bridge and homes on North Cherry Street, and north of Warm Springs Creek.
K	This area is the Fairmont Ranches. It is bounded by the south side of Highway 1 and the Deer Lodge County/Silver Bow County line including Crackerville Road and Fairmont Road.

Source: Bornschein (1994)

Modified Bornschein Sub-Areas Anaconda, Montana



Map 950pa48j
October 16, 1995



occupied room (usually living room or kitchen), and a floor area in the child's bedroom. Water samples were collected from the primary water faucet, normally the kitchen sink. Samples consisted of 100 milliliter (mL) taken immediately upon opening the tap. Water samples were collected in Anaconda only from a subset of 36 homes in subarea A that obtained drinking water from local groundwater.

In Anaconda, soil samples were collected from the yards of 280 homes, and indoor dust was sampled in 278 Anaconda homes. In Opportunity, soil and indoor dust were collected at 20 homes. Three homes in Lost Creek and 2 homes in Fairmont were sampled for soil and indoor dust. Tapwater samples were collected from a total of 61 homes: 36 in Anaconda, 20 in Opportunity, 3 in Lost Creek, and 2 in Fairmont. All of the tapwater samples were collected from homes using groundwater as the source of drinking water.

Soil samples were analyzed using atomic absorption (AA) for arsenic and lead; dust and water samples were only analyzed for arsenic. Method validation included cross checks with inductively coupled plasma-mass spectroscopy (ICP-MS). Analysis methods were consistent with EPA's Test Methods for Evaluating Solid Waste, Volume 1A: Laboratory Manual, Physical/Chemical Methods (EPA 1988a).

Indoor dust was not analyzed for lead. Since lead remains a COPC for soil in the communities, lead contamination of interior dust must be extrapolated from other data. This extrapolation is described in Section 3.3.1, and uncertainties associated with the extrapolation discussed in Section 5.3.3.

Tapwater was analyzed only for arsenic; therefore, it is possible that other contaminants could be present at significant concentrations. For Opportunity, this does not appear to be a significant data gap, since more complete metals analyses of drinking water in Opportunity (CDM Federal 1994a) did not detect any contaminants other than arsenic at significant concentrations. However, Opportunity is more distant from many waste sources and is

characterized by generally lower soil contamination levels than is Anaconda. Thus, it may not be appropriate to extrapolate results of groundwater sampling directly from Opportunity to Anaconda. Lack of data for chemicals other than arsenic in Anaconda domestic groundwater is a potentially important data gap. Possible impacts of this data gap are discussed in Section 5.3.1.

Data Evaluation

Quality control results for the Bornschein data were presented in *Field and Laboratory Quality Control Results* (Roda 1995). A review of this report revealed that the lack of several key pieces of information prevented an assessment of the acceptability and useability of the data and an evaluation of bias and precision of the data. Much of the needed information was provided; however, data that would allow for the quantitative evaluation of data bias and precision were generally not available.

- For water samples, deficiencies included the lack of analysis of a continuing calibration blank and the lack of laboratory duplicates.
- In the interior dust data set, there were no analyses of preparation, method, or digestion blanks, nor was there an analysis of a continuing calibration blank. Laboratory duplicate analyses were conducted; however, it could not be determined whether duplicates were processed through the entire protocol, or if duplicate readings of the same sample were taken at the instrument.
- In the soil sampling investigation, no preparation blanks were prepared to assess whether cross-contamination occurred between samples. Independent reference material was not analyzed to check bias of the analytical system, nor were duplicate samples analyzed to assess precision.
- In the analysis of urine samples, key steps, including the digestion of organic arsenic and the use of an arsenic reducing agent, were not included in the written protocol. For the low standard (60 micrograms per liter [$\mu\text{g/L}$]), the mean reported value was 48 $\mu\text{g/L}$, with a range from 34 to 61 $\mu\text{g/L}$. On a percent recovery basis, the mean would be 80%, with a range from 56.7 to 101.7%. Urine values for arsenic near or below 60 $\mu\text{g/L}$ are most likely biased low.

Evaluation of data quality is, therefore, based only on existing information. The results of this evaluation indicate that data from the environmental and biological samples can be assumed to be equivalent to screening quality data. This data evaluation is described in greater detail in a memorandum from D. Neuman, Reclamation Research Unit - Montana State University, to B. Alexander, CDM Federal, included as Appendix A.

Samples were collected from areas where exposure potential is considered high (i.e., yards and homes) and soil samples were collected from surface soil (0 to 2 cm). Further, a large number of samples is available for most subareas of the study. Thus, sampling protocols were sufficient to adequately characterize the distribution of surface contamination within reasonable exposure units for Opportunity and most of Anaconda. It is expected that data are representative of surface contamination in these communities, and groundwater contamination in subarea A of Anaconda and in Opportunity. Little or no data are available for Galen, Fairmont, and Warm Springs from this study, and no quantitative risk assessment would be appropriate for these areas based on the Bornschein data alone.

A more quantitative analysis of Bornschein data is consistent with the above conclusions. When these data are compared to data collected by PTI (1992 and 1993), there is little difference between the two data sets (Section 2.2). This increases the level of confidence in the soil data that were available.

Summary

The data are of sufficient quality and quantity to be used in a quantitative risk assessment of Anaconda and Opportunity. Data collected in Lost Creek and Fairmont are not of sufficient quantity to be used in a quantitative risk assessment. However, due to the proximity of Fairmont to Opportunity, these samples were combined into one area and referred to as Opportunity.

2.1.2.3 CDM Federal (1994a)

CDM Federal (1994a) collected a total of 20 groundwater samples from residential drinking water wells located in and near Opportunity. Sampled wells were chosen based on their location, depth, accessibility, tap location with respect to any storage tank, filtration device, or water softener, and owner permission. Because surface contamination is likely to affect the shallow portion of an aquifer first, samples were preferentially taken from wells completed less than 100 feet below grade. To the extent possible, samples were collected to provide the best representation of aquifer water quality (i.e., samples were collected before the water passed through any storage tank, filtration device, or water softener).

Samples were analyzed for metals and metalloids by ICP-MS according to EPA Method 200.8 with the exception of iron, which was analyzed according to EPA Method 6020 CLP-M. EPA Method 300.0 was used to analyze samples for sulfate.

Data Evaluation

Data were evaluated for precision, bias, and completeness. This evaluation concluded that all arsenic, copper, zinc, and sulfate data and 20% of the manganese data are enforcement quality. All cadmium, iron, and lead data and 80% of the manganese data were qualified because of elevated blanks and are considered screening quality. All data satisfied the Level B criteria.

Further, samples were collected from areas where exposure potential is high (i.e., where residents use local groundwater to supply drinking water), and sampling was focused on the shallowest wells, which are most likely to be impacted by surface contamination. A significant number of samples was collected from a variety of locations within the community. Given the number of wells sampled, and the relatively homogeneous distribution of surface contamination, it seems unlikely that there may be significant hot spots where

groundwater contamination is significantly higher than suggested in the data. The assumption that data can be used to represent potential groundwater quality in the community of Opportunity is reasonable.

Summary

The quality and quantity of the data collected by CDM Federal from residential wells in the community of Opportunity are sufficient for use in quantitative risk assessment.

2.2 COMPARABILITY OF AVAILABLE DATA

Soil investigations were conducted by PTI (1992 and 1993) and Bornschein (1992 and 1994), and there were initial concerns that soil arsenic concentrations from the two studies were not comparable. Therefore, data from both studies were evaluated statistically to determine:

- (1) If significant differences exist between arsenic concentrations found in surface soil samples collected for these two studies in Anaconda and nearby communities on an area-by-area basis
- (2) If significant differences do occur, whether these differences can be explained by differences in sampling methodology

If the differences between the two data sets were significant, a more extensive evaluation would have to be carried out to determine which data set best represents exposure conditions within the community.

Results of the statistical tests, however, indicate that PTI (1992 and 1993) data and Bornschein (1992 and 1994) data are not significantly different for eight of nine areas compared. Bornschein soil arsenic concentrations for subarea H were significantly greater (two-tailed $p = 0.05$) than PTI soil arsenic concentrations. The statistical findings suggest that the two investigations sampled the same soil "population." Perhaps more importantly, it

seems clear from examination of the analysis that similar results would be obtained regardless of which data set was used in the quantitative risk assessment. Thus, even if there are differences which are not readily detected statistically, they will have no substantive effect on the results of the risk assessment. The statistical analysis of these data sets is described in detail in Appendix B.

It is concluded that using either data set or a combination of the data will result in similar characterization of arsenic in surface soils for Anaconda and nearby communities. However, the Bornschein samples were collected from yards where actual exposure to children may occur. The PTI (1992 and 1993) soil samples were collected both from yards and pasture or idle land. Further, for arsenic, the Bornschein data contains paired soil and interior dust samples which provide a better characterization of the total exposure environment for children in Anaconda. Based on the above, it was concluded that the Bornschein data adequately characterizes concentrations of arsenic and lead in soil for the purposes of this assessment. Thus, the PTI data are not included in the quantitative risk assessment.

2.3 BACKGROUND SAMPLING

If site-specific background concentrations of COPCs are known, incremental risks (i.e., risks due to mine waste over and above any risks associated with exposure to background levels of chemicals) can be estimated. Site-specific background concentrations are defined in this HHRA as concentrations of chemicals present in soil or groundwater but unrelated to past smelting activities.

Background data compiled for the Mill Creek OU RI are suitable for use in this HHRA. For this investigation, background concentrations in soil and groundwater were established based upon available data for several regional Montana communities.

Because of natural variations, background is considered as a distribution and not as a single value and is, therefore, reported as a range that represents the upper and lower 95% confidence interval of the mean.

2.3.1 BACKGROUND SOIL CONCENTRATIONS

Regional background soil samples were collected from non-impacted areas of Helena Valley and the Montana communities of Philipsburg, Townsend, and Livingston. Based on these regional data, ranges of background soil metals concentrations (mg/kg) are:

Arsenic	6 - 16
Lead	18 - 70

Soil concentrations of arsenic found at the site are significantly greater than background concentrations of arsenic in soil. It is unlikely that background concentrations of arsenic contribute significantly to total potential arsenic exposures. Background concentrations of lead in soil are generally less than reported for samples taken from the site. The mean concentration of lead in site soil is 384 mg/kg, compared to the maximum background value of 70 mg/kg. However, the lower end of the range of lead concentrations in site soil (23 - 2,153 mg/kg) overlaps background concentrations of lead in soil. These data suggest that background may not contribute greatly to potential lead exposures in areas where lead concentrations are high enough to present a significant risk. It is unknown whether background concentrations presented include potential contributions from lead-based paint. Background soil samples were collected from regional communities where lead-based paint could potentially be present. The latter is a potentially significant source of lead exposure in communities unrelated to past smelter operations.

2.3.2 BACKGROUND GROUNDWATER CONCENTRATIONS

The Mill Creek OU RI used background groundwater information gathered for the Anaconda Smelter RI/FS. For this study, a literature review and technical evaluation of background wells were conducted and nine background wells were sampled. Based on these data, ranges of background groundwater metals concentrations ($\mu\text{g/L}$) are:

Arsenic	1.6 - 12.6
Lead	0.9 - 5.0

Arsenic was detected infrequently in site samples collected from residential groundwater wells located in Anaconda, Opportunity, Lost Creek, and Fairmont. Concentrations of arsenic in these site groundwater samples, which ranged from non-detect to 13.8 $\mu\text{g/L}$, overlap with background concentrations of arsenic. Background arsenic may contribute significantly to total arsenic exposure via residential groundwater wells onsite.

Lead was not detected in site samples collected from residential groundwater wells in Opportunity.

2.4 SUMMARY OF DATA USED FOR QUANTITATIVE RISK ASSESSMENT

This section presents a brief description of the data used to calculate exposure point concentrations. Section 2.4.1 summarizes the overall data adequacy. Section 2.4.2 discusses the data for site soils, Section 2.4.3 discusses the data for site groundwater, and Section 2.4.4 discusses the data for interior dust.

2.4.1 SUMMARY OF DATA ADEQUACY

The data evaluation identifies sufficient data to support quantitative risk assessment only for the communities of Anaconda and Opportunity. Sampling locations are too widely spread over the rest of the area addressed in this risk assessment to allow quantitative assessment. However, risk-based screening levels for arsenic are developed for receptors located in areas outside of Anaconda and Opportunity (Section 6.0). These screening levels can be used to help evaluate potential risks in these areas. For example, screening levels can be compared to isopleths for arsenic in surface soil to help identify geographic areas, if any, where current or future risks might be of concern. Likewise, screening levels can be compared with any additional data that may become available in the future, i.e., when land is developed, to identify geographic areas where potential risks may be unacceptable.

2.4.2 SOILS

As described above, surface soil samples used to calculate exposure point concentrations are selected from the Bornschein (1992 and 1994) study. In this study, soil and other media were sampled in the communities of Anaconda and Opportunity. Anaconda was separated into several subareas for this investigation to insure variation in potential risks within the town were adequately assessed. Numerous residential yards were sampled within each subarea, and several samples were collected from different locations within each yard, including the perimeter of the house, lawns, play areas, gardens and bare spots. All of these data are considered useful and are included in the exposure point concentrations calculations (Section 3.3.1).

2.4.3 GROUNDWATER

Anaconda

As described above, only the Bornschein study sampled water from the town of Anaconda. This study collected tapwater samples only from homes within subarea A, which use domestic groundwater as their drinking water source. Samples were analyzed only for arsenic. The public water supply of the town of Anaconda, used as the drinking water source for the other subareas, is from an area considered uncontaminated and is not contaminated by arsenic. Uncertainty regarding the presence of contaminants other than arsenic in domestic groundwater is discussed in Section 5.3.1.

Opportunity

Groundwater investigations in the town of Opportunity were conducted by Bornschein (1992 and 1994) and CDM Federal (1994a). Bornschein (1992 and 1994) collected tapwater samples from 20 homes in Opportunity that had private domestic wells. Samples were analyzed only for arsenic. CDM Federal (1994a) collected groundwater samples from 20 domestic wells in Opportunity and analyzed these samples for a suite of metals and metalloids. Only arsenic was detected in concentrations presenting a potential health hazard. Due to the relatively small number of samples available, groundwater data from both the CDM Federal (1994a) and Bornschein (1992 and 1994) studies are used to develop exposure point concentrations for arsenic in domestic groundwater in the town of Opportunity.

2.4.4 DUST

Residents may be exposed to contaminated interior dust in their home. As described above, Bornschein (1992 and 1994) collected a total of 479 interior dust samples from homes in Anaconda, Opportunity, Lost Creek, and Fairmont. These samples were analyzed only for

arsenic. Anaconda was separated into several subareas for this investigation to insure a more representative assessment of potential risks. Data quality and quantity are considered sufficient for calculation of exposure point concentrations.

No data are available for lead in interior dust. Estimates of lead in dust are extrapolated from arsenic data (Bornschein 1992 and 1994) as described in Section 3.3.1.

3.0 EXPOSURE ASSESSMENT

The objectives of this exposure assessment are to identify potential human populations that may be exposed to site-related chemicals, determine the potential pathways through which exposure may occur, and estimate the magnitude, frequency, and duration of potential human exposures. Results of the exposure assessment for arsenic are presented as pathway-specific chronic daily intake (CDIs) for each receptor population. Lead exposures are estimated using the EPA IEUBK Lead Model, Version 0.99. The results of the exposure assessment for lead are presented as estimated blood-lead concentrations, integrated over possible exposure pathways, for each receptor population.

An exposure pathway generally consists of the following four elements:

- A chemical source and mechanism of release
- An environmental retention or transport mechanism for the released chemical
- A point of potential human contact with contaminated media
- A route of exposure (inhalation, ingestion, dermal absorption) at the point of contact

The absence of any one of the elements in an exposure pathway makes that pathway incomplete. No exposure is possible for incomplete pathways, and no CDIs can be calculated.

Section 3.1 describes the site setting, including the physical setting and potentially exposed populations. Section 3.2 identifies complete exposure pathways. Section 3.3 quantifies potential exposures. Section 3.4 compares predicted and measured exposures to arsenic. Section 3.5 discusses uncertainties associated with the exposure assessment.

3.1 CHARACTERIZATION OF SITE SETTING

The following sections characterize the physical setting and potentially exposed human populations within the Anaconda Smelter NPL Site. Discussion is focused on characteristics of the physical setting and of human populations that may influence potential exposures. This information supports the identification of complete exposure pathways in Section 3.2.

3.1.1 PHYSICAL SETTING

The Anaconda Smelter NPL Site is located in southwestern Montana, at the southern end of Deer Lodge Valley, approximately 25 miles northwest of the city of Butte (Figure 1-1). The site covers approximately 200 square miles, primarily in Deer Lodge County. The surface elevation in the study area ranges from 7,200 feet above mean sea level in the southwestern portion of the area to approximately 4,700 feet at its northeast corner.

3.1.1.1 Meteorology

The climate of Anaconda is classified as semi-arid with moderate wind conditions, long, cold winters, and cool summers (ARCO 1991). Climate in the higher mountain elevations is alpine to subalpine (MultiTech 1987). The average annual temperature measured in Anaconda is 43 degrees Fahrenheit (°F). The warmest month based on the 30-year average daily maximum temperature is July (79°F); the coldest month is January (14.5°F) based on the 30-year average daily minimum temperature.

Weather data collected for the period of 1951 to 1980 at the National Climatic Data Center (NCDC) site at East Anaconda (Montana No. 2604, elevation 5,511 feet) indicate the average annual precipitation is approximately 14 inches. The wettest months are May and June averaging 1.9 and 2.3 inches, respectively. The area receives at least 0.1 inch of precipitation

113 days/year. Mean annual snowfall in Anaconda is 63 inches, based on data for 1951 through 1974 (ARCO 1991).

Winds at the top of the smelter's smokestack generally blow at an average speed of about 11 miles per hour (mph), with gusts up to 80 mph (RCG 1995; Taskey 1972). Ground level winds range in speed from 3.6 to 4.1 meters per second (m/sec), or 7.9 to 9.1 mph (Life Systems 1993). Annual wind rose data indicate that prevailing winds are from the south to southeast, with lesser components from the north to northwest and southwest (CDM Inc. 1985).

3.1.1.2 Geology

The Anaconda Smelter NPL Site is located in and near the western margin of the southern Deer Lodge Valley in Deer Lodge County of southwestern Montana. This valley may be described as a north-south trending intermontaine valley bound at its margins by normal faults. The interior of the valley represents a structural downdropped block or graben. The foothills surrounding the valley are composed primarily of bedrock consisting of sedimentary, igneous, and metamorphic rocks of Tertiary to Pre-Cambrian geologic age.

The southern Deer Lodge Valley is predominantly filled with consolidated and semi-consolidated Tertiary sediments derived from weathering and erosion of the surrounding upland areas, and smaller amounts of Tertiary volcanics. Unconsolidated alluvium and glacial outwash deposits of Quaternary geologic age complete the stratigraphic section of valley fill material in the southern Deer Lodge Valley. As Quaternary valley fill deposits become saturated in the broad interior of the valley, they often represent the most prolific water-bearing material at the site.

3.1.1.3 Vegetation

Current vegetative cover is composed primarily of weedy grasses and shrubs, including Great Basin wildrye (*Elymus cinereus*), rabbit brush (*Chrysothamnus spp.*), and horsebrush (*Tetradymia canescens*) (Taskey 1972; ARCO 1991). Aspen (*Populus tremuloides*) are found on sheltered slopes and basins, and juniper (*Juniperus scopulorum* and *J. horizontalis*) are scattered on the hillsides (ARCO 1991; Taskey 1972). Willows (*Salix spp.*) occupy scattered portions of the drainages (ARCO 1991). Barren areas are found in the Anaconda area, including areas of Smelter Hill, Mount Haggin, the Old Works area, portions of Stucky Ridge, the Anaconda Ponds, the Opportunity Ponds, and the entire flatland between Willow Creek and Lost Creek east of Smelter Hill (RCG 1995). Generally, plant cover and diversity increase with distance from the smelter (Taskey 1972).

3.1.1.4 Hydrogeology

The principal aquifers at the site occur in the upper few hundred feet of alluvium, glacial outwash, and valley-fill located beneath the valley floor of the southern Deer Lodge Valley; alluvium and glacial outwash deposits in the Warm Springs Creek and Mill Creek tributary valleys; and in the lowland creek volcanics and Tertiary alluvial fan material underlying the surrounding foothills area. Typically, groundwater yields are highest from Quaternary alluvial and glacial outwash aquifers located beneath the valley floors of the southern Deer Lodge Valley and Warm Springs Creek and Mill Creek tributary valleys. These aquifers typically dominate groundwater use as a domestic and public water supply at the site.

The direction of groundwater flow is generally away from the upland areas of the site toward the central axis of the southern Deer Lodge Valley, then north following the direction of flow of the Clark Fork River. The alluvial aquifer at the site located beneath the southern Deer Lodge Valley is primarily recharged by valley through-flow from alluvial aquifers in the Warm Springs Creek, Mill Creek, and Silver Bow Creek, tributary valleys, in-flow of

groundwater from the surrounding bedrock aquifer through valley sidewalls, inflow from the underlying bedrock aquifer, infiltration of surface water along portions of perennial streams, infiltration of surface water from ponds and lakes, infiltration of surface water from irrigation, and direct infiltration of precipitation.

Depth to groundwater at the site is highly variable. The depth to groundwater below ground surface (bgs) in the alluvial aquifer in the vicinity of the southern Deer Lodge Valley ranges from less than 5 feet to approximately 50 feet. The depth to groundwater in the alluvial/glacial outwash aquifers in the Warm Springs Creek and Mill Creek tributary valleys ranges from approximately 10 feet to greater than 100 feet. Depth to groundwater bgs in the surrounding upland areas at the site range from approximately 25 feet to greater than 150 feet.

3.1.1.5 Hydrology

Five perennial streams, which are part of the Upper Clark Fork River system, are identified in the study area. These streams include Silver Bow Creek, Willow Creek, Mill Creek, Warm Springs Creek, and Lost Creek. The Mill-Willow Bypass, which is also located in the study area, redirects surface water flow from Willow Creek and Mill Creek around the Warm Springs Ponds. The confluence of Warm Springs Creek with discharge from the Warm Springs Ponds and the Mill-Willow Bypass forms the head waters of the Clark Fork River in the northeast portion of the study area.

Flow rates in perennial streams at the Anaconda Smelter NPL Site are typically high in the spring and early summer due to runoff of snowmelt and precipitation. Baseflow conditions typically prevail in late fall and winter.

Groundwater recharge of perennial streams at the site generally occurs along the lowermost reach of each stream, a short distance (1-3 miles) upstream of its confluence with the Clark

Fork River. Loss of surface water flow in perennial streams of the site to the alluvial aquifer typically occurs along their uppermost reach during periods of high flow.

3.1.2 POTENTIALLY EXPOSED POPULATIONS

A mixture of land uses in the study area suggest a variety of potential receptors. The focus of this assessment is on area residents, since data for non-residential areas outside of the communities of Anaconda and Opportunity are sparse and insufficient to support quantitative assessment. However, risk-based screening levels are developed for other receptors and land uses in Section 6.0. Thus, the following discussion includes characterization of current and likely future land uses throughout the study area.

3.1.2.1 Current Site Conditions

According to the *Anaconda-Deer Lodge County (ADLC) Comprehensive Master Plan* (Master Plan) (Peccia & Associates *et al.* 1990), 471,350 acres of the 472,320 total acres of county land area are identified as rural and the remaining 970 acres are urban. Much of the rural land is National Forest land used for conservation and recreational purposes. The majority of privately owned land is agricultural.

There are five communities located in the study area. These include Anaconda and Opportunity, for which risks will be quantitatively evaluated, and Fairmont, Warm Springs, and Galen. Anaconda is the largest community, with a population of approximately 10,000 persons (1990 census data). Anaconda has a public drinking water supply, which draws water from surface water and groundwater sources outside the area of potential impact of past smelter operations. Some homes in the Anaconda area, however, have private groundwater wells. Rural areas such as Opportunity, Warm Springs, Galen, and rural farm residences use groundwater wells to provide drinking water. Available information, however, suggests that contamination of currently used groundwater sources is minimal.

The portion of the Superfund Study Area in commercial and residential use is small, according to the Master Plan. Excluding the communities of Anaconda and Opportunity, the number of occupied housing units within the study area is 115, with the majority within Teresa Ann Terrace and Cedar Park Homes. Commercial uses cited by the Master Plan include the Town Pump store and gas station, a used car lot, and a racquetball club.

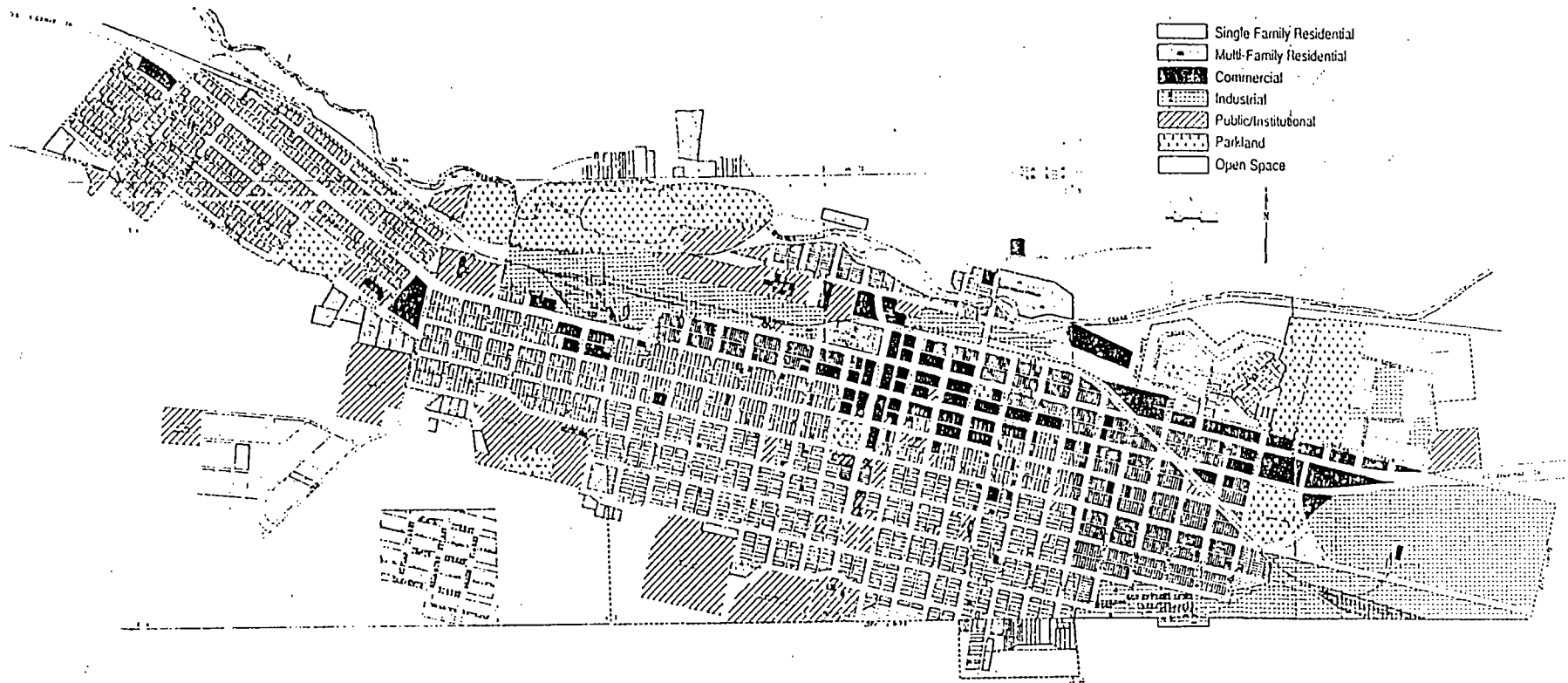
In the community of Anaconda, land is primarily residential, surrounding a commercial core area (Figure 3-1). The Opportunity area is a mixture of large- and small-lot residential with intermingled livestock grazing and mobile homes (Figure 3-2). The Master Plan also states that there are nine businesses located within Opportunity.

Residents of Anaconda and other communities participate in recreational activities such as dirt-bike riding, mountain biking, hiking, hunting, and swimming; these activities may result in exposure to arsenic and/or lead in soils within the study area.

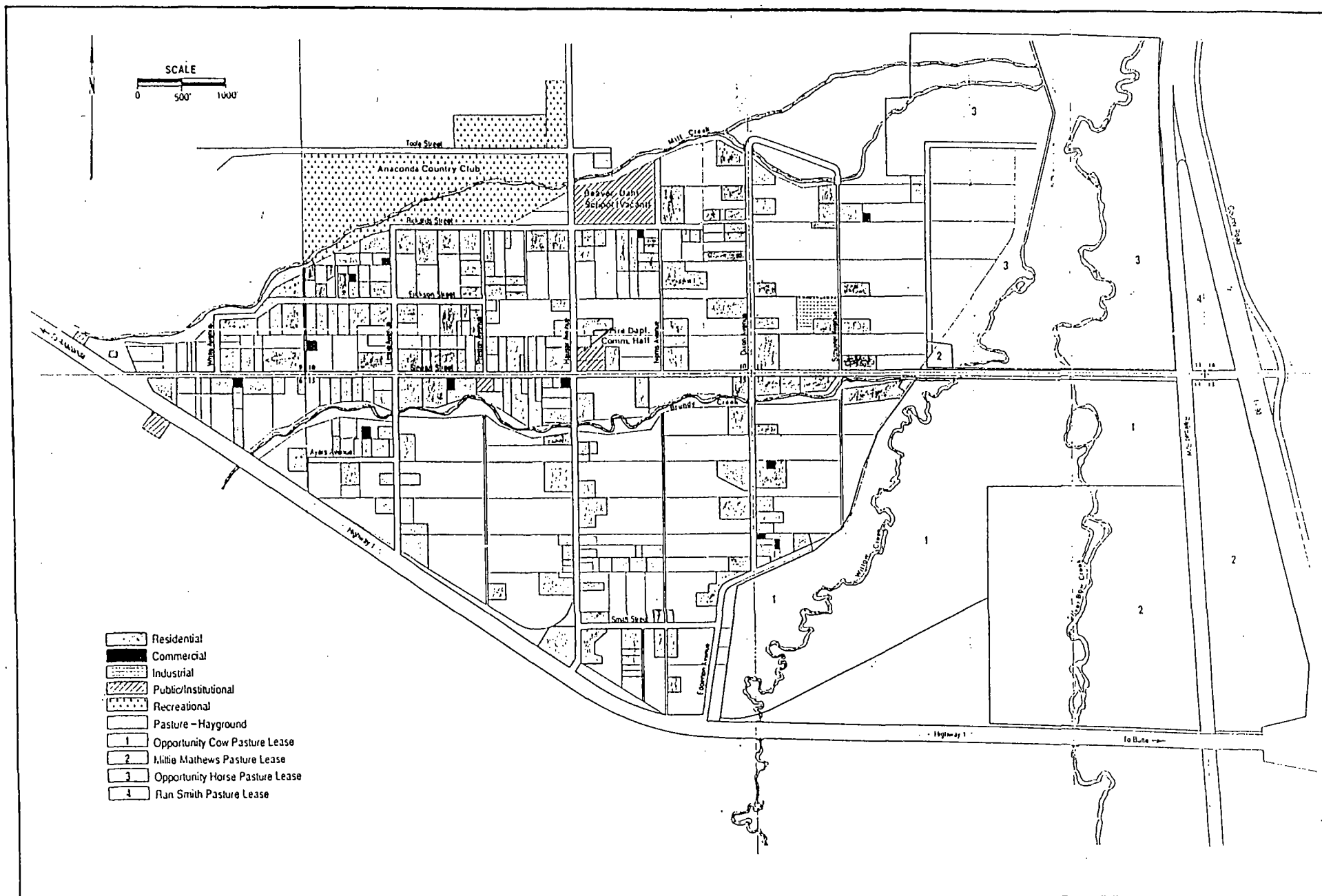
Farms and residences are scattered throughout the site. Approximately 32% of Deer Lodge County is used for agricultural purposes, including crops like spring wheat, barley, oats, hay and potatoes; the average farm size in the county is 2,113 acres (personal communication, Montana Agriculture Line, 8/3/94). It is thought that local consumption of crops is minimal; crops are generally sold and dispersed to a wide range of areas. However, agricultural workers may be exposed directly to arsenic and lead in soils during agricultural activities, such as plowing, planting, field maintenance, and harvesting.

Residents may have private gardens in which fruits and vegetables are grown for personal consumption. Anaconda resident survey responses indicate consumption of locally grown fruits and vegetables is minimal (Bornschein 1993). Livestock production in Deer Lodge County is relatively low compared to other Montana counties; Deer Lodge County ranks as 53 out of 56 counties for beef production (personal communication, Montana Agriculture Line, 8/3/94). Farms may have cattle, sheep, and hogs; however, there are typically no more

FIGURE 3-1
Land Use in Anaconda



Land Use in Opportunity



than 2 animals per farm. Chickens are raised on most farms in the area. It is estimated that consumption of locally raised beef is low, as the majority of cattle raised are sold out of state (personal communication, Montana Agriculture Line, 8/3/94). Based on this information, it appears that exposure to arsenic and lead through the ingestion of local contaminated livestock is negligible. This assumption is supported by Anaconda resident survey responses (Bornschein 1993), which also indicated negligible consumption of locally grown livestock. Moreover, analyses in the Streamside Tailings (SST) OU HHRA (CDM Inc. 1994) indicates that, even if local livestock are consumed, exposure to arsenic and lead through this pathway is not expected to be significant due to the minimal concentrations sequestered in tissue.

3.1.2.2 Future Site Conditions

In the future, areas of the site that are currently undeveloped could be developed for a variety of purposes, including recreational, commercial, residential, or agricultural. Also, lands that are currently used for agricultural purposes could be developed for other uses, such as residential. Risk-based screening levels have been developed for such exposures.

In Anaconda, Opportunity, and the Superfund area, it is anticipated that county regulations and permit requirements described in the *ADLC Development Permit System* (ADLC DPS) (Peccia & Assoc. 1992) will facilitate the protection of Superfund remedies, and require future implementation of Superfund remedies when development occurs. The ADLC DPS was developed to carry out the policies of the Master Plan and is expected to be the primary public regulatory mechanism for controlling development in the Superfund and adjacent areas. It will set forth permissible arsenic levels for each land use (recreational, commercial, occupational, and residential) and a system for permitting based on these levels. Development would be prohibited unless a site has been, or as part of the development, will be remediated to protect human health. Also, the regulations adopted for the Superfund area are expected to protect sites with soil caps, barriers, or other structures from destruction through any proposed development. Limitations on development in sensitive areas, such as

wetlands, floodplains, in stream corridors, or in steep slopes, will also apply in the Superfund area.

Presently, permissible risk-based arsenic screening levels for recreational and commercial/occupational land uses have been established and incorporated into the ADLC DPS. These levels were derived in the *OW/EADA Baseline Risk Assessment* (Life Systems 1993). A permissible arsenic level for residential land use will be established as one outcome of this risk assessment.

3.1.2.3 Subpopulations of Concern

Subpopulations of concern are sensitive receptor populations who might be particularly susceptible to chemical exposure. They may include infants, the elderly, or individuals with respiratory problems depending on the COPCs and the nature of the exposures. Often exposure points for sensitive receptors include hospitals, nursing homes, schools, and day care centers. For this HHRA, sensitive receptors for exposures to lead have been identified as children ages 0 to 6 years. Children in this age range tend to have higher soil ingestion than older children and adults, and are more sensitive to adverse effects from exposure to lead.

3.1.2.4 Summary

Based on current and future land uses described above, the following populations are considered most likely to be exposed to arsenic and lead at the Anaconda Smelter NPL Site:

- Current and future residents
- Agricultural workers
- Recreational users
- Commercial workers

As described in Section 2, arsenic and lead concentrations in soil and groundwater outside of the towns of Anaconda and Opportunity are not adequately characterized due to the relatively small number of samples collected. Therefore, only risks to current residents of Anaconda and Opportunity are assessed quantitatively in this HHRA. Risk-based screening levels are, however, developed (Section 6.0) for other receptors.

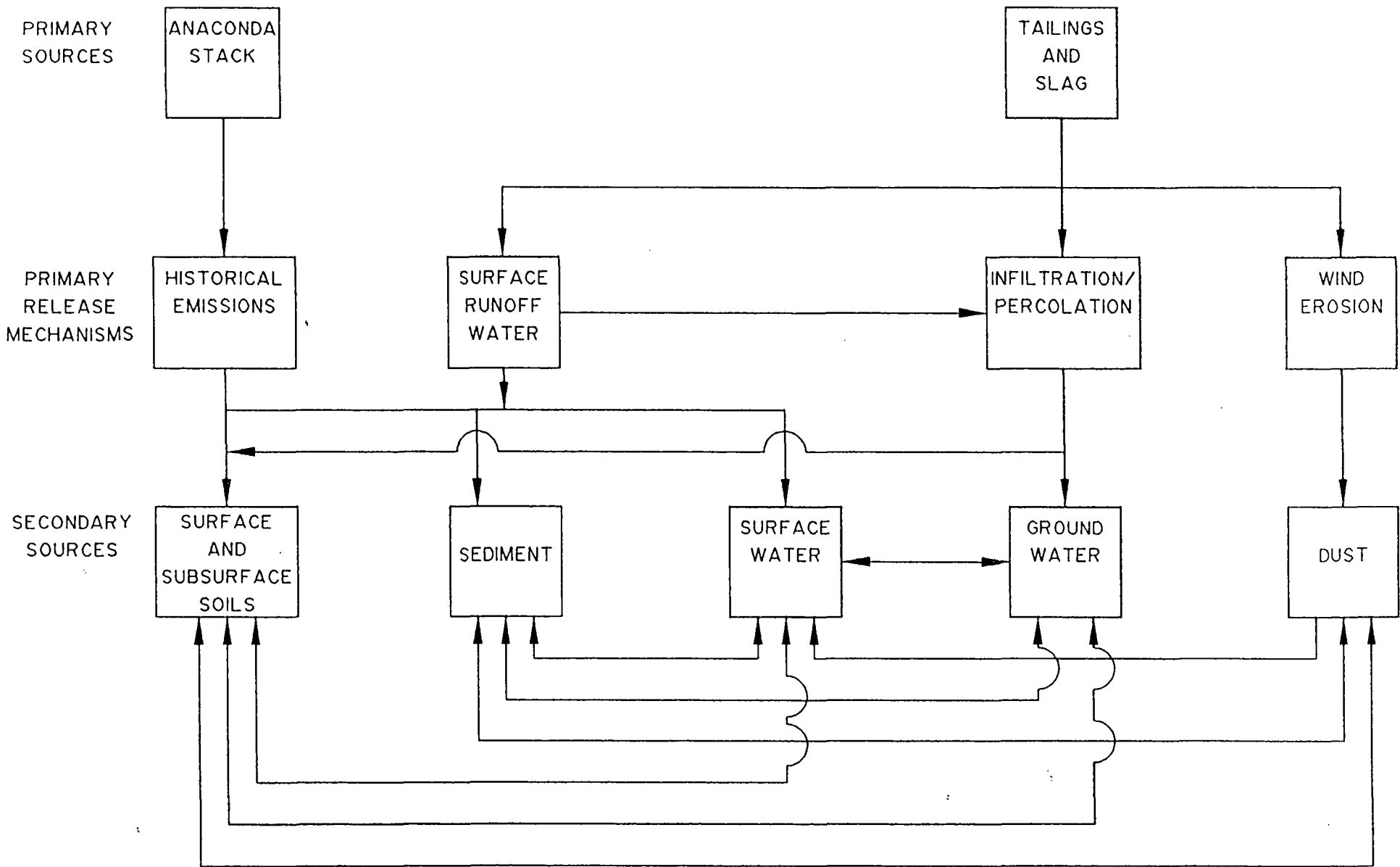
3.2 IDENTIFICATION OF EXPOSURE PATHWAYS

The SCEM (Figure 3-3) for the Anaconda Smelter NPL Site presents primary sources of contamination, primary release mechanisms, secondary and tertiary sources of contamination, and potential human receptors. The SCEM presents reasonable pathways of exposure from primary sources of contamination to potential receptors. Each of these pathways is discussed below.

The two primary sources of contamination within the study area are historical air emissions from the Anaconda Smelter stack and from the tailings and slag remaining from the smelting process. Materials released from the stack were small particulates not captured by the emission controls in place at the plant. The primary release mechanism for tailings and slag is wind erosion, although some release via infiltration/percolation, and runoff has also occurred. Contamination in air emissions was/is transported via dry or wet deposition from the air into three secondary sources, soil, surface water, and sediment.

Contamination in the tailings and slag was also transported via infiltration/percolation into two secondary sources, soils and groundwater. In fact, some tailings are currently in contact with shallow groundwater. Finally, contamination in the tailings and slag is transported via runoff water into three secondary sources, soil, surface water, and sediment. The location of tailings and slag sources suggest that runoff would carry contaminants into sediments of nearby intermittent streams, and into permanent streams only indirectly.

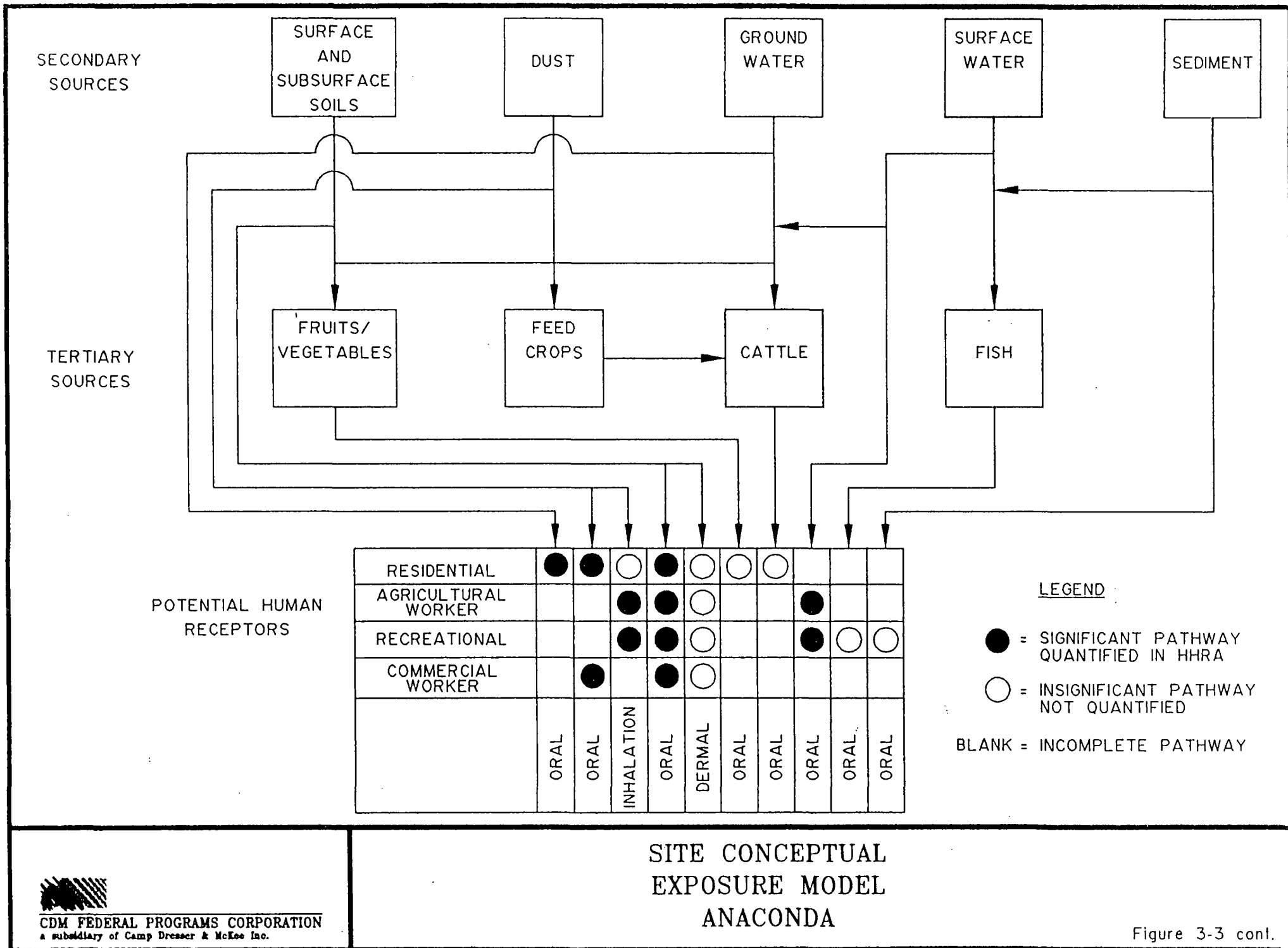
313



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SITE CONCEPTUAL EXPOSURE MODEL ANACONDA

Figure 3-3



Transport of contaminants can occur among the secondary sources. Contaminants in surface soils can be transported to surface water (via runoff), groundwater (via infiltration/percolation), and fugitive dust (via wind). Contaminants in sediment can be transported to surface water (via desorption/dissolution), groundwater (via infiltration/percolation), and fugitive dust (from a intermittent stream bed via wind). Contaminants in groundwater can be transported to sediment (via groundwater flow), surface water (via groundwater flow), and soils (via groundwater flow or use of groundwater as irrigation water). Surface water can transport contaminants to groundwater (via infiltration/percolation), sediment (via sorption/precipitation), and surface soils (via runoff or use of surface water as irrigation water). Contaminants in fugitive dust can be transported to surface water (via dry or wet deposition) and surface soils (via dry or wet deposition).

Contaminants may also move from secondary sources to tertiary sources. Tertiary sources include vegetables/fruits or crops, cattle, and fish. Crops can uptake contaminants from soil, absorb contaminants from surface water or groundwater, or absorb contaminants (via leaf) in fugitive dust. Cattle can ingest contaminants from crops, soil, sediment, groundwater, or surface water, or inhale contaminants in fugitive dust. Fish can ingest contaminants in sediment or surface water, or absorb contaminants from surface water.

Ingestion of Arsenic and Lead in Contaminated Surface Soil

Soils may have been contaminated by airborne historic smelter emissions, re-entrainment of contaminated dust, and runoff and erosion from waste piles. Children and adults typically ingest small amounts of soil through hand-to-mouth contact. This pathway is considered complete for residents, agricultural workers, recreational users (dirt bikers), and commercial workers. Estimates of potential exposure via this pathway to residents of Anaconda and Opportunity are presented in this HHRA. All other receptors are evaluated using risk-based screening levels (Section 6.0).

Ingestion of Arsenic and Lead Contaminated Interior Dust

Contaminants in soil may be transported indoors in air, tracked in on shoes, carried in on clothes or pets fur, etc. Once indoors, soil particles become part of interior dust. Children and adults typically ingest small amounts of dust through hand-to-mouth contact. This pathway is considered complete for residents and commercial workers. Estimates of potential exposure to residents of Anaconda and Opportunity are presented in this HHRA. Commercial workers are evaluated using risk-based screening levels (Section 6.0).

Inhalation of Arsenic and Lead in Contaminated Soil and Waste Particles in Air

Arsenic and lead forms at this site are not volatile; however, they may be released to the air from contaminated soils, dust, and waste piles as respirable particulate matter by wind or mechanical disturbances. Monitoring data indicate that levels of arsenic and lead in air are below current regulatory limits (Life Systems 1993). Therefore, inhalation of particulate matter released by wind erosion is not assessed quantitatively for residents or commercial workers. However, persons engaged in activities that result in the short-term release of large quantities of dust (i.e., farming, dirt-bike riding) may be exposed to high concentrations of arsenic and/or lead via inhalation of dust. Data are insufficient to quantify such exposures, however, for most areas within the scope of this HHRA. Therefore, agricultural and recreational exposure scenarios are evaluated in Section 6.0 through the use of risk-based screening levels.

Indirect Exposure to Arsenic and Lead through Ingestion of Fruits/Vegetables Grown in Contaminated Soil

Current or future residents might grow fruits and vegetables in soil contaminated by surface runoff from waste piles and/or by deposition of contaminated dust. These fruits and vegetables may take up chemicals from the soil into the edible portion of the plant. However,

as described in the HHRA for the Mine Flooding OU, Silver Bow Creek/Butte NPL Site. exposure through this pathway is not expected to contribute significantly to site-related risks, and is, therefore, not further evaluated in this risk assessment.

Indirect Exposure to Arsenic and Lead through Ingestion of Contaminated Meats

Current or future residents might raise livestock, such as cattle. Livestock could, in theory, take up arsenic and/or lead from ingested soil and food and sequester these chemicals in their tissues. However, information presented in the SST OU HHRA indicates that this is unlikely to occur in significant amounts. Additionally, local residents consume little locally grown livestock. Exposure through this pathway is expected to be negligible for arsenic and lead even where local livestock is consumed based on information presented in Section 3.1.2.1. This pathway is not likely to significantly contribute to site-related risks and is, therefore, not further assessed in this HHRA.

Indirect Exposure to Arsenic and Lead through Ingestion of Contaminated Fish

Several creeks flow through the Anaconda Smelter NPL Site (Section 3.1.1.5). Fish in these creeks can ingest arsenic and lead in sediment or surface water, or absorb arsenic and lead from surface water. Current or future residents and recreational users could be exposed through ingestion of these fish. Although this is a plausible exposure pathway, screening level calculations presented in Life Systems (1993) indicate that risks resulting from fish ingestion would be very low. Therefore, exposure from ingestion of contaminated fish is not evaluated further in this HHRA.

Ingestion of Arsenic in Contaminated Groundwater

Current residents in Anaconda and Opportunity use surface water and groundwater as sources of drinking water. Residents could be exposed if these drinking water sources are

contaminated by arsenic. Exposure through this pathway is evaluated in the risk assessment for residents in Anaconda and Opportunity.

Ingestion of Arsenic and Lead in Contaminated Surface Water

Surface water runoff following rainstorms and snowmelt has led to contamination of surface water and sediment in site creeks. Area residents might visit affected creeks and be exposed to surface water through incidental ingestion during wading and other water play activities. Exposure to arsenic and lead might also occur through aquatic recreation in a small pool near Opportunity Ponds. It seems unlikely that such exposures would be associated with significant risks, based on the results of the risk assessment recently completed for the SST OU of the Silver Bow Creek NPL Site (CDM Inc. 1994). Surface water and sediment are heavily contaminated with arsenic and metals in this OU, yet even conservative estimates of potential exposures were not associated with significant risk. Risk-based screening concentrations are developed in Section 6.0 for this exposure pathway.

Dermal Exposure to Arsenic and Lead in Contaminated Surface Water

Only recreational users of surface water within the study area would be potentially exposed through this pathway. Lead is not expected to be significantly absorbed across the skin and several risk assessments performed for sites within the Clark Fork Basin have concluded that dermal exposure is insignificant for this metal. Dissolved arsenic in surface water may, however, be absorbed to some extent, although significant exposures are not expected based on results of the SST OU risk assessment (CDM Inc. 1994). This pathway is addressed using risk-based screening levels developed in Section 6.0.

Ingestion of Arsenic and Lead in Contaminated Sediment

Potential exposures for recreational visitors could occur during visits to surface water in the study area. Incidental ingestion of sediments might occur in much the same way as incidental ingestion of soils. However, recreational visitors are assumed to spend the majority of their time in the water, where sediments are not expected to adhere to skin. Additionally, visitors are assumed to bathe following swimming. Therefore, this HHRA assumes that contact with sediments would be minimal. This pathway is, therefore, considered insignificant and exposure to recreational users is not quantified in this HHRA.

Dermal Exposure to Arsenic and Lead in Contaminated Soil

It is expected that residents, recreational users, agricultural workers, and commercial workers might have dermal contact with contaminated soil. Only limited data are available on the rate at which metals cross the skin into the blood from soil or dust particles; therefore, dermal exposure to metals was not included in the quantitative assessment. It is not considered likely that omission of this pathway causes a significant underestimate of risk because uptake of metals across the skin, especially from soil, is generally believed to be minor.

Summary

From the information above, pathways of concern are:

Exposure Pathways for Residents (Adults and Children Ages 0 - 6):

- Ingestion of surface soils
- Ingestion of interior dust
- Ingestion of groundwater

Agricultural Workers (Adults):

- Ingestion of surface soils
- Inhalation of dust

Recreational Users (Dirt Bike Riders):

- Ingestion of surface soils
- Inhalation of dust

Recreational Visitors (Swimmers):

- Ingestion of surface water
- Dermal exposure to surface water

Commercial Workers (Adults):

- Ingestion of surface soils
- Ingestion of interior dust

3.3 QUANTIFICATION OF EXPOSURE

The amount of a chemical that receptor populations take into their bodies following exposure is referred to as chemical intake. The CDI is expressed in units of milligrams of chemical per kilogram of body weight per day (mg/kg-day) and is the standard expression of long-term daily exposure. Intake depends on the exposure point concentration of chemicals in a medium (e.g., groundwater), and exposure assumptions specific to the receptor population, including how often and how long the exposure occurs (exposure frequency and duration), body weight, and contact rate. Depending on the exposure route, contact rate is equivalent to the volume of food, water, or soil ingested, air inhaled, or surface water contacted dermally each day. The period of time over which exposure is averaged, or the averaging time, is used to convert total intake into daily intake.

Section 3.3.1 describes the estimation of exposure point concentrations. Pathway-specific and general exposure assumptions (e.g., frequency and duration of exposure) are provided in Section 3.3.2.

3.3.1 ESTIMATION OF EXPOSURE POINT CONCENTRATIONS

Exposure point concentrations are estimates of the level of a COPC in a medium at the exposure point. The approaches used in this HHRA to calculate exposure point concentrations for different media are as follows:

3.3.1.1 Soils

As described above, only Bornschein (1992 and 1994) soil sampling data are used to evaluate risks. In this study, Anaconda was separated into subareas (A, B, C, D, E, F, I, and J) to better characterize possible differences in exposure conditions within the community. For this assessment, subarea F, the subarea closest to Smelter Hill, was subdivided into areas F1 and F2 to ensure that potential exposures in this area were adequately addressed (Figure 2-1). Opportunity was retained as a separate study area (subarea G). Numerous yards within each subarea were sampled and soil was collected from several locations within each yard, including play, house perimeter, garden, hardpack, and bare areas. Soil concentrations for arsenic and lead from all of these samples were averaged for each yard¹. An exposure point concentration for arsenic was derived for each area by calculating the 95% upper confidence limit (UCL) of the mean (EPA 1992b) of the arithmetic average soil concentrations for each residence assuming lognormal distribution. Use of the 95% UCL provides reasonable confidence that the average concentration will not be underestimated. Arsenic exposure point concentrations for soils of each subarea are shown in Table 3-1.

Lead intake is evaluated by the IEUBK Lead Model, Version 0.99. Average lead concentrations in soils of each subarea, rather than the 95% UCL of the mean, are used as lead exposure point concentrations (Table 3-2).

¹ Averaging of all soil samples for each yard will tend to minimize any contribution of lead paint that is present.

TABLE 3-1
Anaconda Summary Statistics for Soil Data (Arsenic mg/Kg)

Sample Area	Sample Number	Geometric Mean	Arithmetic Mean	Ln STD	Minimum Detection	Maximum Detection	95th UCL
Area A	44	82.27	86.92	0.34	38.40	171.20	95.76
Area B	60	130.84	138.97	0.35	59.33	229.80	150.52
Area C	17	183.46	191.43	0.30	107.50	306.33	221.65
Area D	11	214.86	225.26	0.34	136.00	340.00	282.23
Area E	47	190.57	195.31	0.22	92.00	292.50	206.31
Area F1	52	237.46	246.36	0.28	126.50	409.25	264.60
Area F2	36	190.57	204.30	0.39	82.50	373.50	231.64
Area I*	3	109.73	117.13	0.45	67.50	165.50	830.91
Area J	10	132.95	140.66	0.36	64.00	193.60	181.24
OPPORTUNITY	22	122.73	127.56	0.30	128.90	219.25	145.05

*Area I should use max detect because of limited sample number (3)

TABLE 3-2

Summary of Soil Lead Data

Exposure Area	Number of Residences	Minimum Concentration (mg/kg)	Maximum Concentration (mg/kg)	Average Concentration (mg/kg)	Standard Deviation
A	44	19.80	312.00	75.92	54.42
B	60	44.60	1183.00	256.65	215.04
C	17	57.20	851.00	476.49	245.23
D	11	110.20	812.50	419.37	230.53
E	47	110.00	1388.00	581.66	282.04
F1	52	111.00	2152.70	533.99	302.75
F2	36	60.00	1220.20	508.14	288.65
I	3	60.50	87.00	75.03	13.44
J	10	14.30	303.20	191.20	88.43
Opportunity	22	46.20	351.20	133.98	81.85
ALL AREAS	302	14.30	2152.70	364.03	297.24

3.3.1.2 Dust

The dust data for arsenic were provided in the Bornschein (1992 and 1994) study. Interior dust samples were collected from homes in Anaconda and Opportunity. Three interior dust samples were commonly taken for each residence. Data from a single residence were averaged prior to calculation of exposure point concentrations. Statistical tests of dust arsenic concentrations suggest a lognormal distribution. Therefore, data were logtransformed and arsenic exposure point concentrations derived for each subarea by calculating the 95% UCL of the arithmetic mean (EPA 1992b) for the lognormalized data (Table 3-3).

Dust samples were not analyzed for lead. However, analysis of paired soil and interior dust measurements for arsenic suggest a transfer coefficient of 0.43 for movement from soil to dust (Bornschein 1994). That is, concentrations of arsenic in indoor dust are about 43 percent of arsenic concentrations in outdoor soil. This value is derived from multiple regression analyses performed by Bornschein (1994) on data collected during the Anaconda arsenic exposure study, and represents information from paired soil and dust samples from over 300 locations. If arsenic and lead in smelter wastes are assumed to move in similar fashion from soil into dust, then the arsenic coefficient can be used to estimate transfer of lead.

In addition to a transfer coefficient, it is expected that there will be a "background" level of lead in homes unrelated to outdoor soil. This background may be represented by the y-intercept of the soil/dust regression line. Since no dust lead measurements have been made in Anaconda, the y-intercept must be obtained indirectly, again using the arsenic data. If arsenic is used as a surrogate for lead, a constant term for lead can be calculated assuming that the ratio of the constant term for average soil lead to indoor dust lead is the same as the parallel ratio for arsenic.

The approach used to estimate the above ratio for arsenic in this assessment follows that recommended by EPA (1995e). This guidance suggests that a "y-intercept", or constant term,

TABLE 3-3
 Anaconda Summary Statistics for Dust Data (Arsenic mg/Kg)

Sample Area	Sample Number	Geometric Mean	Arithmetic Mean	Ln STD	Minimum Detection	Maximum Detection	95th UCL
Area A	44	50.40	57.68	0.51	17.80	153.40	66.54
Area B	60	57.97	66.10	0.50	19.30	284.40	74.20
Area C	17	46.53	56.84	0.64	14.90	157.10	79.90
Area D	11	121.51	130.07	0.38	67.00	277.90	166.09
Area E	47	83.93	91.20	0.39	37.00	237.90	100.44
Area F1	51	116.75	130.74	0.49	29.00	393.60	149.90
Area F2	36	95.58	106.17	0.48	19.70	224.40	125.13
Area I*	3	44.26	54.30	0.77	23.10	103.40	16228.33
Area J	9	52.98	57.79	0.47	20.65	95.87	86.72
OPPORTUNITY	22	75.19	83.07	0.45	33.30	205.40	100.76

*Area I should use max detect because of limited sample number (3)

can be estimated as the average of measured values that are less than some given percentage of the mean. It is found that only four measurements of arsenic in indoor dust contain arsenic at concentrations of 25 percent of the mean or less. These four measurements are thus taken to indicate "background" for indoor arsenic in dust. The average of these four measurements is about 18 mg/kg. Since the average arsenic concentration in outdoor soil for the same data set is 175 mg/kg, the constant term for arsenic is 10 percent of the site-wide average outdoor soil level.

Extrapolating measurements of arsenic in soil and dust to lead results in the following equation for estimating interior lead concentrations.

$$PB_{dust} = (0.43 \times PB_{soil}) + (0.1 \times \text{mean } PB_{soil})$$

Where: PB_{dust} = the interior dust lead concentration (mg/kg)
 PB_{soil} = the average subarea soil lead concentration (mg/kg)
mean PB_{soil} = the community-wide average lead concentration for Anaconda

Since the sitewide average lead concentration in outdoor soil is 364 mg/kg, the constant term for lead is estimated to be about 36 mg/kg. Interior dust lead concentrations for the different subareas are shown in Table 3-4. Use of the above equation assumes that transport of arsenic and lead from soil to dust is similar, and ignores any potential contribution from interior lead-based paint. This adds uncertainty to estimates of lead exposure within the study area.

3.3.1.3 Groundwater

Bornschein (1992 and 1994a) sampled tapwater in subarea A of Anaconda, from homes using groundwater as their source of drinking water, and in the town of Opportunity. CDM Federal (1994a) sampled domestic groundwater wells in Opportunity.

TABLE 3-4

Calculated Concentrations of Lead in Indoor Dust

Subarea	(mg/kg)
A	69.0
B	146.8
C	241.3
D	216.7
E	286.5
F1	266.1
F2	254.9
I	68.7
J	118.6
Opportunity	94.0
All Areas	192.9

Samples collected by Bornschein (1992 and 1994) were analyzed only for arsenic. Samples were not analyzed for lead. Lead was not detected in samples collected by CDM Federal (1994a). Therefore, default values for lead in drinking water were used in the IEUBK model.

Separate arsenic exposure point concentrations were developed for Anaconda and Opportunity drinking water (Table 3-5). The exposure point concentration for drinking water is the 95% UCL of the mean (EPA 1992b) for all of the arsenic concentrations measured in drinking water of subarea A and Opportunity. Although it is unlikely that individuals would be exposed to drinking water from multiple domestic wells, wells were combined to provide total population risk for the subareas. An arsenic concentration of nondetect was used for subareas in Anaconda which obtain drinking water from the public water supply. It should be noted that the large number of non-detects and low detections, especially in Opportunity, make it difficult to determine the appropriate data distribution. Thus, the default, using normal statistics, was used in accordance with EPA guidance (EPA 1992b).

3.3.1.4 Data Manipulation

When a chemical was detected in a medium (i.e., surface soils), samples having no detection for that chemical in the same medium were treated as if they contained the chemical at one-half the detection limit. Further, if the chemical is present in a sample below the sample quantitative limit (SQL), EPA recommends using one-half the SQL as a proxy concentration. This methodology follows recommendations in EPA's *Risk Assessment Guidance for Superfund, Part A* (1989a), which suggests that concentrations most representative of potential exposure at a site will consider both positively detected results and non-detected results within appropriate exposure units.

TABLE 3-5

**Arsenic Exposure Point Concentrations
in Groundwater - Anaconda Smelter NPL Site**

AREA	Sample Number	FOD	Min (ug/L)	MAX (ug/L)	Arithmetic Mean (ug/L)	95th ¹ UCL (ug/L)
ANACONDA	36	92%(33/36)	1.1	9.9	2.46	2.91
OPPORTUNITY	42	21%(9/42)	1.1	13.8	1.73	2.45

¹Because of the low frequency of detection (FOD) for the Opportunity data (21%), the data are neither normal or lognormally distributed. The distribution of the Anaconda data is rather flat. The 95th percent upper confidence limit of the arithmetic mean (UCL), used as the exposure point concentration, was calculated with the student-T normal distribution formula, shown below.

UCL= mean + t(standard deviation / square root of sample number).

3.3.2 EXPOSURE ASSUMPTIONS

Pathway-specific CDIs are estimated using exposure point concentrations and exposure assumptions specific to the receptor population. Generally, exposure assumptions are selected so that their combination results in an estimate of the reasonable maximum exposure (RME) for that pathway (EPA 1989a). RME is defined as an exposure well above the average but still within the range of those possible. Recent guidance (EPA 1992c) suggests that the RME should fall in the range of the 90th to the 99.9th percentile of possible exposures.

It is expected that different people will have different levels of contact with contaminated media, therefore, a range of exposures is also provided by estimating the central tendency exposure (CTE) for each exposure pathway. CTE uses exposure assumptions that predict an average or best estimate exposure to an individual. The presentation of risks for both RME and CTE scenarios provides the risk manager a range of risk for the site.

In keeping with EPA risk assessment guidelines (EPA 1989a), exposure assumptions derived from site-specific data are used when available, so that risks can be evaluated on a case-by-case basis. Where site-specific data are unavailable, standard EPA default assumptions for both RME and CTE exposures are used in accordance with EPA guidance (EPA 1989a, 1989b, 1991b, 1993a). The following is a discussion of exposure assumptions used to quantify intakes for the residential scenario.

Exposure assumptions are summarized in Tables 3-6 to 3-9.

3.3.2.1 Exposure Assumptions Common to All Pathways

The RME uses upper range estimates for some, but not all, exposure assumptions so that their combination results in a reasonable upper range estimate of exposure for that pathway. On the other hand, CTE uses best estimates for most exposure assumptions to estimate exposures

TABLE 3-6
VARIABLES COMMON TO ALL EXPOSURE EQUATIONS
ANACONDA SMELTER SITE
CENTRAL TENDENCY EXPOSURE

Variable Symbol	Variable Definition	Variable Values That Could Be Selected	Variable Value Selected/Percentile	Reason for Variable Selection	Reference
BW	Body Weight	0 - 80 kg			
	Adult (residential)	67 - 72 kg	70 kg	Recommended by EPA	EPA 1993b
	Child (residential, age 0-6 years)	11 to 17 kg	15 kg	Recommended by EPA	EPA 1993b
AT	Averaging Time				
	Carcinogenic	365 days x 70 years	365 days x 70 years	Recommended by EPA	EPA 1989a
	Noncarcinogenic	Varies with ED	365 days x ED	Recommended by EPA	EPA 1989a
ED	Exposure Duration				
	Adult (residential)	0 - 70 years			
	Carcinogenic		7 years	Recommended by EPA	EPA 1993b
	Noncarcinogenic		7 years	Recommended by EPA	EPA 1993b
	Child (residential, age 0-6 years)	0 - 6 years			
	Carcinogenic		2 years	Recommended by EPA	EPA 1993b
	Noncarcinogenic		2 years	Recommended by EPA	EPA 1993b

TABLE 3-7
VARIABLES COMMON TO ALL EXPOSURE EQUATIONS
ANA CONDA SMELTER SITE
REASONABLE MAXIMUM EXPOSURE

Variable Symbol	Variable Definition	Variable Values That Could Be Selected	Variable Value Selected/Percentile	Reason for Variable Selection	Reference
BW	Body Weight	0 - 80 kg			
	Adult (residential)	67 - 72 kg	70 kg	Recommended by EPA	EPA 1993b
	Child (residential, age 0-6 years)	11 to 17 kg	15 kg	Recommended by EPA	EPA 1993b
AT	Averaging Time				
	Carcinogenic	365 days x 70 years	365 days x 70 years	Recommended by EPA	EPA 1989a
	Noncarcinogenic	Varies with ED	365 days x ED	Recommended by EPA	EPA 1989a
ED	Exposure Duration				
	Adult (residential)	0 - 70 years			
	Carcinogenic		24 years	Recommended by EPA	EPA 1993b
	Noncarcinogenic		24 years	Recommended by EPA	EPA 1993b
	Child (residential, age 0-6 years)	0 - 6 years			
	Carcinogenic		6 years	Recommended by EPA	EPA 1993b
	Noncarcinogenic		6 years	Recommended by EPA	EPA 1993b

TABLE 3-8
VARIABLES ASSOCIATED WITH SPECIFIC PATHWAYS
ANA CONDA SMELTER SITE
CENTRAL TENDENCY EXPOSURE

Variable Symbol	Variable Definition	Range of Variable Values	Variable Value Selected	Reason for Variable Selection	Reference
WATER					
IR - Used for Ingestion of Groundwater					
	Ingestion Rate	0 - 2.8 L/day			
	Adult (residential)	0.69-2.8L/day	1 L/day	Recommended by EPA	EPA 1993b
	Child (residential, age 0-6 years)	0.69 - 0.93 L/day	0.7 L/day	Consistent with CTE	
BAF - Bioavailability of Arsenic in Water					
	BAF of Arsenic in Water	0 - 100%	100% percent	Site-specific data	EPA 1995
EF - Used for Ingestion of Groundwater					
	Exposure Frequency	0 - 365 days/yr			
	Adult (residential)	0 -365 days/yr	234 days/yr	Recommended by EPA	EPA 1993b
	Child (residential, age 0-6 years)	0 -365 days/yr	234 days/yr	Recommended by EPA	EPA 1993b
SOIL AND DUST					
IR - Used for Ingestion of Surface Soil and Dust					
	Ingestion Rate	0 - 480 mg/day			
	Adult (residential)	0 - 480 mg/day	50 mg/day	Recommended by EPA	EPA 1993b
	Child (residential, age 0-6 years)	0 - 480 mg/day	100 mg/day	Recommended by EPA	EPA 1993b
FI - Used for Ingestion of Surface Soil					
	Fraction Ingested from Source	0 - 100%			
	Adult (residential)	0 - 100%	45%		
	Child (residential, age 0-6 years)	0 - 100%	45%		
BAF - Bioavailability of Arsenic in Soil					
	BAF of Arsenic in Soil	0 - 100%	18.30%	Site-specific data	EPA 1995
FI - Used for Ingestion of Interior Dust					
	Fraction Ingested from Source	0 - 100%			
	Adult (residential)	0 - 100%	55%		
	Child (residential, age 0-6 years)	0 - 100%	55%		
BAF - Bioavailability of Arsenic in Dust					
	BAF of Arsenic in Dust	0 - 100%	25.80%	Site-specific data	EPA 1995
EF - Used for Ingestion of Surface Soil and Interior Dust					
	Exposure Frequency	0 - 365 days/yr			
	Adult (residential)	0 -365 days/yr	350 days/yr	Recommended by EPA	EPA 1992a
	Child (residential, age 0-6 years)	0 -365 days/yr	350 days/yr	Recommended by EPA	EPA 1992a

TABLE 3-9
VARIABLES ASSOCIATED WITH SPECIFIC PATHWAYS
ANA CONDA SMELTER SITE
REASONABLE MAXIMUM EXPOSURE

Variable Symbol	Variable Definition	Range of Variable Values	Variable Value Selected	Reason for Variable Selection	Reference
WATER					
IR - Used for Ingestion of Groundwater					
	Ingestion Rate	0 - 2.8 L/day			
	Adult (residential)	0.69-2.8L/day	2 L/day	Recommended by EPA	EPA 1993b
	Child (residential, age 0-6 years)	0.69 - 0.93 L/day	1 L/day	Recommended by EPA	EPA 1989b
EF - Used for Ingestion of Groundwater					
	Exposure Frequency	0 - 365 days/yr			
	Adult (residential)	0-365 days/yr	350 days/yr	Recommended by EPA	EPA 1993b
	Child (residential, age 0-6 years)	0-365 days/yr	350 days/yr	Recommended by EPA	EPA 1993b
SOIL AND DUST					
IR - Used for Ingestion of Surface Soil and Dust					
	Ingestion Rate	0 - 480 mg/day			
	Adult (residential)	0 - 480 mg/day	100 mg/day	Recommended by EPA	EPA 1993b
	Child (residential, age 0-6 years)	0 - 480 mg/day	200 mg/day	Recommended by EPA	EPA 1993b
FI - Used for Ingestion of Surface Soil					
	Fraction Ingested from Source	0 - 100%			
	Adult (residential)	0 - 100%	45%		
	Child (residential, age 0-6 years)	0 - 100%	45%		
BAF - Bioavailability of Arsenic in Soil					
	BAF of Arsenic in Soil	0 - 100%	18.30%	Site-specific data	EPA 1995
FI - Used for Ingestion of Interior Dust					
	Fraction Ingested from Source	0 - 100%			
	Adult (residential)	0 - 100%	55%		
	Child (residential, age 0-6 years)	0 - 100%	55%		
BAF - Bioavailability of Arsenic in Dust					
	BAF of Arsenic in Dust	0 - 100%	25.80%	Site-specific data	EPA 1995
EF - Used for Ingestion of Surface Soil and Interior Dust					
	Exposure Frequency	0 - 365 days/yr			
	Adult (residential)	0-365 days/yr	350 days/yr	Recommended by EPA	EPA 1992a
	Child (residential, age 0-6 years)	0-365 days/yr	350 days/yr	Recommended by EPA	EPA 1992a

near the average of the possible range. Some assumptions are, however, common to both RME and CTE estimates. The following exposure assumptions are used for all CDI calculations. Table 3-6 presents the CTE assumptions common to all pathways, and RME assumptions are presented in Table 3-7.

Body Weights

For adult residents, the value selected for body weight (BW) is 70 kg. This is the representative mean BW for men and women between the ages of 18 and 75 (EPA 1993a). For child residents, a BW of 15 kg is used (EPA 1993a).

Averaging Time

Averaging time (AT) is the period in days over which chemical intakes are averaged. For noncarcinogenic chemicals, intakes are averaged over the exposure duration (ED) ($ED \times 365$ days). For carcinogens, intake calculations average the total cumulative dose over a lifetime (assumed to be 25,550 days for a 70-year lifetime). Averaging times differ for carcinogens and noncarcinogens, because the effects of carcinogenic chemicals are assumed to have no threshold. Therefore, any exposure to a carcinogen carries a finite risk of causing cancer during the lifetime of the individual. Within reason, this means that a single large exposure to a carcinogen is expected to carry the same risk as the same total dose divided up into many small exposures. It is therefore most convenient to express intakes of carcinogens in terms of lifetime exposures, regardless of the actual exposure duration (EPA 1989a).

Exposure Frequency

For the soil and dust ingestion pathway, an exposure frequency (EF) of 350 days/year is used as the amount of time a person is at home for both the RME and CTE scenarios (EPA 1993a). This value assumes that a person spends part or all of each day of the year at home,

except for 15 vacation days and is considered appropriate for the soil ingestion pathway (EPA 1993a). For groundwater ingestion, an EF of 350 days/year is used for the RME scenario (EPA 1993a). For the CTE scenario, an EF of 234 days/year is used. This corresponds to the fraction of time estimated that is actually spent at home for both men and women (64%) (EPA 1993a).

Exposure Duration

EDs of 24 and 6 years are used for adult and child RME estimates, respectively (EPA 1993a). This results in ATs for adult and child noncarcinogenic exposures of 8,760 days and 2,190 days, respectively. For the CTE exposure duration, 7 and 2 years are used for adult and child residents, respectively, resulting in ATs for adult and child noncarcinogenic exposures of 2,555 and 730 days, respectively (EPA 1993a).

3.3.2.2 Site-Specific Exposure Assumptions

Pathway specific assumptions for CTE and RME exposures are described below, and presented in Tables 3-8 and 3-9, respectively.

Assumptions for Bioavailability of Arsenic from Soils and Dust

EPA has used the available data to derive site-specific arsenic bioavailability estimates for ingested soil and dust (EPA 1994a, 1995a). These data describe the arsenic concentrations in blood, urine, and feces collected from Cynomolgus monkeys exposed to arsenic by intravenous injection, gavage, and oral administration of capsules containing soil or dust collected in Community Soils OU (Battelle 1994). Arsenic bioavailability was measured as the ratio of the area under the curve (AUC) for arsenic in urine and blood following administration of soil or dust capsules, compared to that following intravenous administration adjusted for the difference in the size of the dose.

Mean absolute bioavailability estimates derived from urine arsenic concentrations were 91%, 18.3% and 25.8%, respectively, for gavage, soil, and dust. Absolute bioavailability estimates derived from blood arsenic concentrations were similar and ranged between 91% and 100% for gavage, 11% and 18% for soil ingestion and 8% and 11% for dust. The results of this study demonstrate that the absorption of arsenic from soils and dust is significantly less than absorption of soluble arsenic from water. Further, the results confirm the previous assumption of nearly complete absorption of ingested dissolved arsenic.

Results from the bioavailability study have been extensively reviewed and are thought to be reasonable site-specific estimates for human bioavailability. They are used directly in this assessment, as follows:

- 25.8% bioavailability for dust
- 18.3% bioavailability for soil
- 100% bioavailability for water

Further discussion of the derivation of these values can be found in an EPA (1995a) memorandum from C. Weis (EPA) to C. Coleman (EPA), describing the EPA review of the Battelle Columbus report: *Determination of the bioavailability of soluble Arsenic and Arsenic in soil and dust impacted by Smelter Activities following oral administration in Cyanomolgus monkeys. Amended Final Report.* This memorandum is included as Appendix C.

Selection of Soil and Dust Ingestion Rate for Children Living in Anaconda

A week-long measurement of soil and dust ingestion in 64 children living in Anaconda was performed by Dr. Edward Calabrese. Using a single "best tracer" methodology, the soil and dust ingestion rate median was 51 mg/day, the mean was 117 mg/day, and the 90th percentile was 277 mg/day. The "four best tracers" study resulted in an ingestion rate median of 39 mg/day, a mean of 83 mg/day and a 90th percentile of 273 mg/day. The findings in the Anaconda soil and dust ingestion study support the Superfund Program's usual approach of

assuming ingestion of 100 mg soil and dust per day as a CTE assumption and 200 mg soil and dust per day as a RME assumption for soil and dust ingestion rates (IRs) of children age 0 - 6 years. Though default assumptions are used for soil and dust IRs for children, these assumptions are clearly consistent with the available site-specific data.

3.3.2.3 Standard Default Exposure Assumptions

Site-specific data are unavailable for several exposure parameters. Default assumptions for these parameters are described below.

Ingestion of Surface Soils and Dust

Fraction Ingested

The IR values used for adults and children include both soil and interior dust. The fraction ingested (FI) values correct for the relative amount of soil or dust ingested. It was assumed for both adults and children that of the total soil and dust ingested, 55% derives from indoor dust and 45% from soil. An assumption for fractionating dose between soil and dust is necessary since (1) indoor dust and soil arsenic and lead concentrations are not the same at exposure points, (2) different bioavailability estimates are used for dust and soil for arsenic, and (3) many studies have found a significant contribution of indoor dust to exposure.

Adult Soil and Dust Ingestion Rate

The RME value selected for the IR for surface soils, soil-like material, and indoor dust for adult residents is 100 mg/day (EPA 1993a). The CTE value selected for IR for adult residents is 50 mg/day (EPA 1993a). This estimate is fractionated between soil and dust using the FI described above.

Ingestion of Water

Adult Water Ingestion Rate

The RME value selected for the IR for water for the adult resident is 2 L/day (EPA 1993b). This is close to the 90th percentile of values measured and/or estimated for the general population in the U.S., and is also the value currently used to establish drinking water standards by EPA's Office of Water (EPA 1993c). The CTE value selected for the IR of water for the adult resident is 1.4 L/day (EPA 1993b). This is based on the average intake observed from five studies in which the observed range was from 0.26 to 2.8 L/day (EPA 1993b).

Child Water Ingestion Rate

For child residents, the RME value selected for the IR of water is 1 L/day (EPA 1989b). The CTE value selected for the IR of water for child residents is 0.7 L/day. This estimate is in keeping with the definition of a CTE value, and is based on professional judgement.

IEUBK Model Default Values

Default values provided in the IEUBK model are used for the following input parameters:

- Lead bioavailability
- Lead concentrations in groundwater
- Lead concentrations in air
- Lead dietary intake
- Maternal blood lead contribution

Model default values are shown in Table 3-10.

TABLE 3-10

Default Model Parameters

AIR CONCENTRATION: 0.100 µg Pb/m ³				SOIL AND DUST		
Indoor Air Pb Concentration: 30.0% of outdoor				SOIL: Constant Concentration		
Other Air Parameters:				DUST: Constant Concentration		
Age (Years)	Time Outdoors (Hours)	Vent. Rate (m ³ /day)	Lung Abs. (%)	Age (Years)	Soil (µg Pb/g)	House Dust (µg Pb/g)
0-1	1.0	2.0	32.0	0-1	Site Specific	Site Specific
1-2	2.0	3.0	32.0	1-2	Site Specific	Site Specific
2-3	3.0	5.0	32.0	2-3	Site Specific	Site Specific
3-4	4.0	5.0	32.0	3-4	Site Specific	Site Specific
4-5	4.0	5.0	32.0	4-5	Site Specific	Site Specific
5-6	4.0	7.0	32.0	5-6	Site Specific	Site Specific
6-7	4.0	7.0	32.0	6-7	Site Specific	Site Specific
DIET: Age (Years) 0-1 1-2 2-3 3-4 4-5 5-6 6-7 (µg/day) 5.53 5.78 6.49 6.24 6.01 6.34 7.00				PAINT INTAKE: 0.00 µg Pb/day		
DRINKING WATER CONCENTRATION: 4.00 µg/L WATER Consumption: Age (Years) 0-1 1-2 2-3 3-4 4-5 5-6 6-7 (L/d) 0.20 0.5 0.52 0.53 0.55 0.58 0.59				MATERNAL CONTRIBUTION: Infant Model Maternal Blood Concentration: 2.50 µg Pb/dL		
ABSORPTION METHODOLOGY: Non-linear Active-Passive						

^a Default values are shown, but site-specific values are used in the analysis.

A non-default value was used for the geometric standard deviation (GSD) in the IEUBK model. The GSD represents the variability of individual blood-lead levels with respect to the geometric mean blood level predicted by the IEUBK model. An inter-individual or individual GSD represents the variability remaining after environmental and age variability have been taken into account. The default GSD value of 1.6 is based on calculations of GSDs from specific sites ranging from 1.3 to 1.8 (Marcus 1992). A GSD of 1.4 was used in the model; this GSD was calculated from site-specific data on children at the Sandy City, Utah Smelter Site (EPA 1995a) and the Bingham Creek, Utah Site (EPA 1995b). The lead contamination originates from smelting activities at the Sandy City site and from historic mining operations at the Bingham Creek site. The lead concentrations in these soils, for the most part, appear to have a fairly homogenous and predictable distribution pattern. The Anaconda Smelter NPL Site is similar in that the lead in soil is distributed in a pattern which is consistent with the smelting activities and seems relatively homogenous within each of the gradients across the site. For this reason, it is reasonable to extrapolate the GSD from the site-specific data of the Sandy City Smelter Site and the Bingham Creek, Utah Site to the Anaconda Smelter NPL Site.

3.3.3 CALCULATION OF CHEMICAL INTAKES — RESIDENTIAL RECEPTOR

Chemical intakes by potentially exposed residential receptors are calculated using exposure point concentrations for each exposure area and the exposure assumptions described above. CDIs are estimated for each selected exposure pathway. In Section 5, CDIs are compared to toxicity values to quantify carcinogenic risks and noncarcinogenic hazards for each exposure pathway evaluated.

SOILS AND INTERIOR DUST

3.3.3.1 Ingestion of Chemicals in Surface Soil and Interior Dust

To determine CDIs associated with incidental ingestion of arsenic in surface soils and interior dust, the following equation is used (EPA 1989a).

$$CDI \text{ (mg/kg-day)} = \frac{CS \times IR \times CF \times FI \times EF \times ED \times BAF}{BW \times AT}$$

Where:	CDI	=	Chronic Daily Intake ((mg/kg)/day)
	CS	=	Chemical Concentration in Soil or Dust (mg/kg)
	IR	=	Ingestion Rate (mg/day)
	CF	=	Conversion Factor (10^{-6} kg/mg)
	FI	=	Fraction Ingested from Contaminated Source (unitless)
	EF	=	Exposure Frequency (days/year)
	ED	=	Exposure Duration (years)
	BAF	=	Bioavailability Factor for COPC in Soil or Dust (unitless)
	BW	=	Body Weight (kg)
	AT	=	Averaging Time (days)

Example Calculations (Soil Ingestion, RME):

$$(NC): CDI ((mg/kg)/day) = \left[\frac{CS(mg/kg) * 200(mg/d) * 10^{-6}(kg/mg) * 0.45 * 350(d/yr) * 6(yr) * 0.183}{15(kg) * (365(d/yr) * 6(yr))} \right] + \left[\frac{CS(mg/kg) * 100(mg/d) * 10^{-6}(kg/mg) * 0.45 * 350(d/yr) * 24(yr) * 0.183}{70(kg) * (365(d/yr) * 24(yr))} \right]$$

$$(C): CDI ((mg/kg)/day) = \left[\frac{CS(mg/kg) * 200(mg/d) * 10^{-6}(kg/mg) * 0.45 * 350(d/yr) * 6(yr) * 0.183}{15(kg) * (365(d/yr) * 70(yr))} \right] + \left[\frac{CS(mg/kg) * 100(mg/d) * 10^{-6}(kg/mg) * 0.45 * 350(d/yr) * 24(yr) * 0.183}{70(kg) * (365(d/yr) * 70(yr))} \right]$$

Where: NC = Noncarcinogenic
C = Carcinogenic

Table 3-11 presents CDIs calculated for ingestion of arsenic in soil and interior dust. Both RME and CTE estimates are provided.

WATER

3.3.3.2 Ingestion of Water

To determine CDI for ingestion of arsenic in contaminated drinking water, the following equation is used (EPA 1989a).

$$CDI ((mg/kg)/day) = \frac{CW \times IR \times CF \times EF \times ED \times BAF}{BW \times AT}$$

Where: CDI = Chronic Daily Intake ((mg/kg)/day)
CW = Chemical Concentration in Water (micrograms/liter, or µg/L)
IR = Ingestion Rate (liters/day)

TABLE 3-11
CHRONIC DAILY INTAKES
INGESTION OF ARSENIC IN GROUNDWATER, SOIL, AND DUST
RME AND CTE RESIDENTIAL SCENARIO
ANACONDA SMELTER SITE
(mg/kg-day)

SUBAREA	GROUNDWATER INGESTION				SOIL AND DUST INGESTION			
	Noncarcinogenic CDI		Carcinogenic CDI		Noncarcinogenic CDI		Carcinogenic CDI	
	(mg/kg-day)		(mg/kg-day)		(mg/kg-day)		(mg/kg-day)	
	RME	CTE	RME	CTE	RME	CTE	RME	CTE
Subarea A	1.01E-04	4.01E-05	2.51E-05	2.63E-06	6.33E-05	3.38E-05	1.03E-05	1.63E-06
Subarea B	0	0	0	0	8.37E-05	4.48E-05	1.36E-05	2.15E-06
Subarea C	0	0	0	0	1.08E-04	5.78E-05	1.76E-05	2.78E-06
Subarea D	0	0	0	0	1.71E-04	9.14E-05	2.78E-05	4.39E-06
Subarea E	0	0	0	0	1.14E-04	6.10E-05	1.86E-05	2.93E-06
Subarea F1	0	0	0	0	1.57E-04	8.41E-05	2.56E-05	4.04E-06
Subarea F2	0	0	0	0	1.35E-04	7.19E-05	2.19E-05	3.46E-06
Subarea I	0	0	0	0	1.03E-04	5.53E-05	1.68E-05	2.66E-06
Subarea J	0	0	0	0	9.95E-05	5.32E-05	1.62E-05	2.56E-06
Opportunity	8.50E-05	3.37E-05	2.11E-05	2.21E-06	9.59E-05	5.13E-05	1.56E-05	2.46E-06

CF	=	Conversion Factor (10^{-3} $\mu\text{g}/\text{mg}$)
EF	=	Exposure Frequency (days/year)
ED	=	Exposure Duration (years)
BAF	=	Bioavailability Factor for COPC in water (unitless)
BW	=	Body Weight (kg)
AT	=	Averaging Time (days)

Example Calculations (Ingestion of Water, RME):

$$NC: CDI ((\text{mg}/\text{kg})/\text{day}) = \left[\frac{CW(\text{ug}/\text{L}) * 1(\text{L}/\text{d}) * 10^{-3}(\text{ug}/\text{mg}) * 350(\text{d}/\text{yr}) * 6(\text{yr}) * 1}{15(\text{kg}) * (365(\text{d}/\text{yr}) * 6(\text{yr}))} \right] +$$

$$\left[\frac{CW(\text{ug}/\text{L}) * 2(\text{L}/\text{d}) * 10^{-3}(\text{ug}/\text{mg}) * 350(\text{d}/\text{yr}) * 24(\text{yr}) * 1}{70(\text{kg}) * (365(\text{d}/\text{yr}) * 24(\text{yr}))} \right]$$

$$C: CDI ((\text{mg}/\text{kg})/\text{day}) = \left[\frac{CW(\text{ug}/\text{L}) * 1(\text{L}/\text{d}) * 10^{-3}(\text{ug}/\text{mg}) * 350(\text{d}/\text{yr}) * 6(\text{yr}) * 1}{15(\text{kg}) * (365(\text{d}/\text{yr}) * 70(\text{yr}))} \right] +$$

$$\left[\frac{CW(\text{ug}/\text{L}) * 2(\text{L}/\text{d}) * 10^{-3}(\text{ug}/\text{mg}) * 350(\text{d}/\text{yr}) * 24(\text{yr}) * 1}{70(\text{kg}) * (365(\text{d}/\text{yr}) * 70(\text{yr}))} \right]$$

Table 3-11 presents CDIs for arsenic calculated for ingestion of arsenic in drinking water.

3.4 COMPARISON OF PREDICTED AND MEASURED EXPOSURES TO ARSENIC

When site-specific measurements of exposure are available, comparison of observed versus predicted exposures may be the best means to evaluate uncertainties in exposure calculations. Where exposure measurements have been carefully made, and can be assumed to be representative, observed and predicted exposures should be in reasonable agreement. Since careful and representative observations of arsenic exposure have been made for children in the communities of Anaconda and Opportunity (Bornschein 1994), a comparison of observed and

predicted exposures has been carried out for children living in these communities. Methodologies used to perform this comparison are presented in Appendix D, *Evaluation of the Data Collected in the Town of Anaconda Using EPA Risk Assessment Methodology* (CDM Federal 1995).

Exposure comparisons were based on observed and predicted urinary arsenic. To support this comparison, arsenic absorption and urinary excretion were assumed to be in equilibrium. This seems a reasonable assumption since children in the study had been living in the same exposure conditions (same residence) for several months to several years prior to the study. In addition, bioavailability estimates for arsenic in soil and dust were based on EPA (1994a, 1995a) analyses of data collected by Battelle (1994). The Battelle study measured arsenic in blood, urine and feces of monkeys following administration of soluble arsenic by injection and gavage, and soils and dust from Anaconda in capsules. These data form the basis for estimation of absolute bioavailability as described in Section 3.3.2.2.

Estimates for daily urine output were taken from measurements made by Bornschein (1994), and presented in Appendix D. For children less than 36 months of age, urine production was estimated to be 240 ml/day. Values for children 36 to 60 months and greater than 60 months were 355 and 432 ml/day, respectively.

Total daily absorption of arsenic was estimated as follows:

$$ABS = \frac{((C_s * 0.45 * BAF_s) + (C_d * 0.55 * BAF_d)) * IR_s * CF_s * EF}{AT} + \frac{C_w * CF_w * IR_w * EF * BAF_w}{AT}$$

All variables are defined in Sections 3.3.3.1 and 3.3.3.2, Tables 3-6 to 3-9, and are also provided in Appendix D.

Urinary excretion was estimated from absorption estimates using:

$$EXC = \frac{ABS * CF_{abs}}{RATE * CF_{exe}}$$

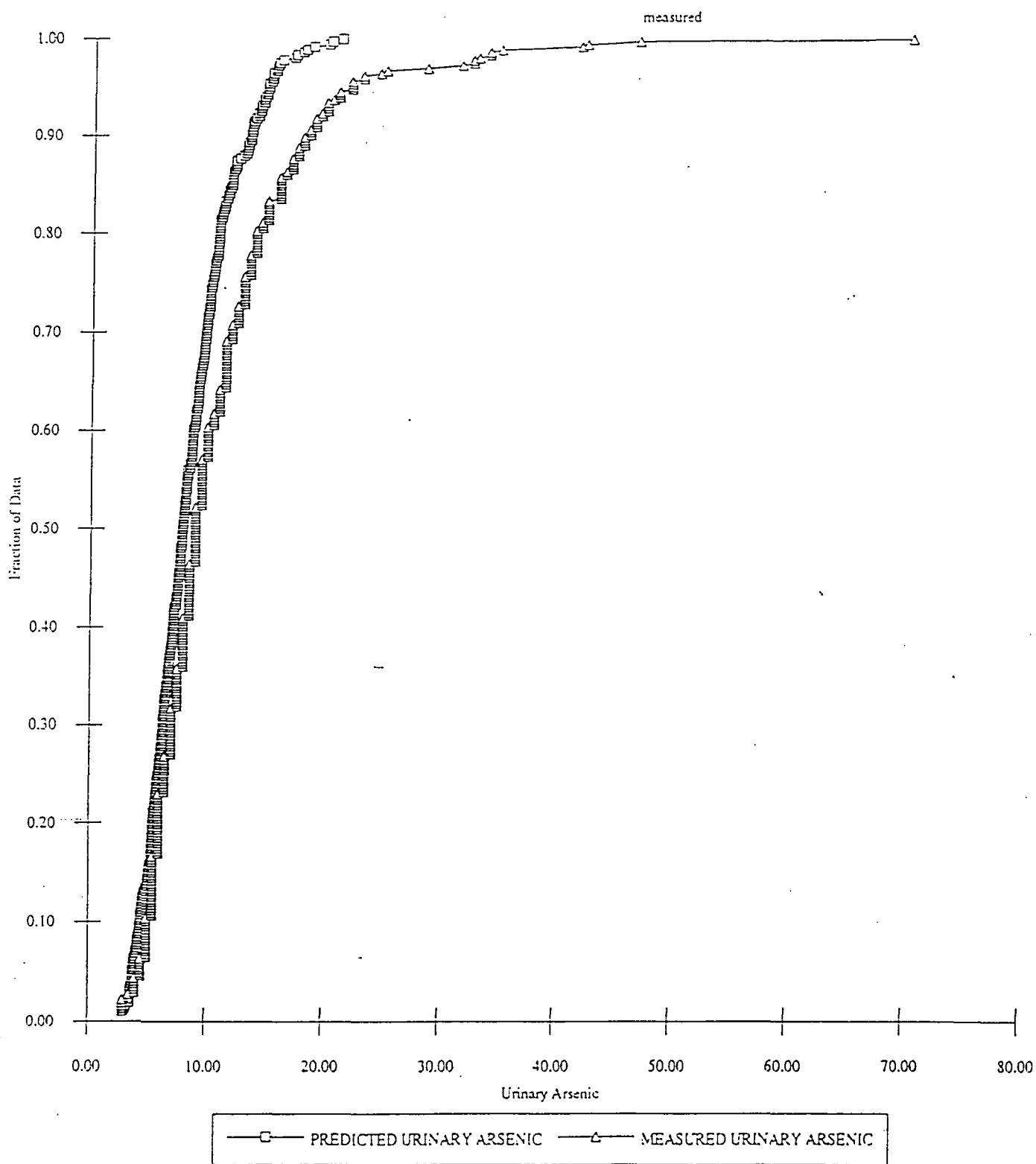
Where:

EXC	=	Urinary arsenic excretion (µg/L)
ABS	=	Estimated daily arsenic absorption (mg/d)
Cf _{abs}	=	Conversion factor (10 ³ µg/mg)
RATE	=	Estimated urinary output (ml/d, varies with age)
Cf _{exe}	=	Conversion factor (10 ⁻³ L/ml)

Observed and predicted urinary arsenic, based on speciated arsenic measured by Bornschein, are presented in Figure 3-4. Kruskal-Wallis one-way analysis of variance does not eliminate the null hypothesis that populations of observed and predicted urinary arsenic levels have the same mean. However, it is apparent that calculated urinary arsenic underpredicts measured values when urinary arsenic concentrations exceed 10 µg/L.

Predicted urinary arsenic levels are based on exposure assumptions intended to represent central tendencies in exposures. Where observed exposures, as reflected in urinary arsenic values, are relatively low, these CTE estimates seem to reproduce measured exposures well. At higher observed exposures, CTE evaluation tends to underpredict exposures. Where arsenic in urine was measured at 20 µg/L, for example, the predicted value was 15.5, about 23 percent lower. Where arsenic was measured at 29 µg/L, the predicted value was about 45 percent lower (16 µg/L). The single child who had a measured arsenic level of 80 µg/L was predicted to excrete only about 20 µg/L. Thus, at the highest measured exposure, predicted values based on CTE could be low by a factor of four. This is currently the best estimate for uncertainty in calculations of arsenic exposure in the most exposed individuals.

FIGURE 3-4
MEASURED AND PREDICTED SPECIATED URINARY ARSENIC



SPLAQRPH.XLS Chart 2
7/13/95 11:02 AM

Final Baseline HHRA
CDM FEDERAL PROGRAMS CORPORATION
I.R. ANAC 006/HHRA_FNL.ANA/011096

3.5 UNCERTAINTIES ASSOCIATED WITH EXPOSURE ASSESSMENT

The exposure assessment relies on assumptions for a variety of exposure parameters.

Assumptions used are variously based on:

- Site-specific information
- EPA guidance
- Professional judgement

Exposure scenarios and assumptions used in this risk assessment are also based on discussions with EPA Region VIII personnel.

The combination of exposure assumptions used in the assessment is expected to provide realistic estimates for exposure of individuals living in Anaconda and Opportunity. As discussed in Section 3.4, urinary arsenic levels predicted from exposure assumptions used in this assessment, and those measured by Bornschein (1994), are in reasonable agreement for exposures based on central tendency. This increases confidence in exposure calculations for this assessment.

It should be noted, however, that the comparison of observed and predicted exposures can only be made for children. This risk assessment estimates exposure for longer durations including substantial periods of adulthood. Uncertainties in exposures for adults may therefore be important in defining the overall uncertainty in exposure estimates. For example, little information is available for evaluating soil/dust ingestion rates in adults. If default values for adults (50 and 100 mg/d for CTE and RME respectively), are substantially in error, total predicted exposures would be similarly under- or overestimated. Thus, it is not possible to evaluate some major sources of uncertainty using the comparison with Bornschein data.

Choices made for adult exposure parameters are within the ranges suggested by EPA and should be conservative for assessing adult exposure. For example, adults are likely to ingest

less soil than young children because of reduction in hand-to-mouth behavior with age. Adult ingestion rates in this assessment are half those suggested for young children. If this assumption underestimates exposures, it is likely to do so by a factor of less than two. It seems unlikely that soil/dust ingestion rate assumptions for adults result in significant underestimation of exposure.

Frequency and duration of exposure are also important determinants of exposure which may not be adequately addressed in the comparison of observed and predicted urinary arsenic levels in children. Much of the exposure for arsenic is anticipated to occur during adulthood. Adults, because of work and other out-of-home activities, may be exposed less frequently than young children. In addition, exposure duration is an important component of exposure evaluation, yet there is no site-specific information on residence times for Anaconda and Opportunity. If residence times in these communities are significantly different than national norms, exposures may be under- or overestimated.

Exposure frequency is estimated at the high end of those possible, allowing only for a two week per year vacation. Many individuals may spend more time than this away from home and/or may spend limited time at home on most weekdays because of work commitments. These individuals may receive less exposure than that estimated in this assessment. However, it is expected that significant numbers of individuals (for example non-working parents) will spend significant amounts of time each day at their homes. Moreover, many individuals will work close to their residences and thus be in a similar exposure environment both at home and at work. The high exposure frequency used in this assessment is not expected to be appropriate for all individuals in the potentially exposed population. However, it is assumed that a high exposure frequency is reasonable for a large fraction of the population and that this is not a major source of uncertainty in the risk estimates.

Exposure duration can have a significant impact on exposure estimates. National norms suggest that the 90th percentile for time at one residence is about 30 years, and that average

residence time is about 9 years. If the population in Anaconda and Opportunity is either more sedentary or more mobile than the nation as a whole, risks could be either under- or overestimated.

In many cases, small rural communities have many residents that stay in the community for long periods of time. It would not be surprising to see upper range estimates of a few decades for time at one residence. In addition, it is likely that people in the community may move within the community (e.g., from an apartment to a private residence). Anaconda has, however, seen a net loss in population since the closure of the smelter in 1980. This may suggest that residence times are dropping as children who grow up in the area, or adult residents, leave to find jobs, go to school, etc. It is, thus, difficult to determine how Anaconda and Opportunity may compare to national norms.

It seems unlikely, however, that uncertainties in exposure duration would be of great significance. For example, if an more reasonable upper range estimate for time at one residence was either 20 or 40 years, RME estimates would go down or up by only 33 percent. Anaconda and Opportunity would have to be very different from the national norms to greatly affect exposure calculations.

It should be noted that predicted blood lead levels based on lead exposure of children have not been confirmed through direct observations of blood lead levels. Default values were used in the IEUBK model for lead bioavailability, lead concentration in groundwater, air, and food, and maternal blood lead contribution. There is uncertainty associated with the default value for lead absorption from soil. The IEUBK model uses a default value of 30%, however, gastrointestinal absorption of lead has been reported to range from 5 to 72% depending upon soil geochemistry, species of lead, and age, nutritional status, and state of health of the exposed individual. In the absence of site-specific data regarding soil lead absorption, the IEUBK default value is considered the most acceptable for use.

A non-default value was used for the GSD in the IEUBK model; this value was calculated from site-specific data on children at the Sandy City, Utah Smelter site (EPA 1995a) and the Bingham Creek, Utah Site (EPA 1995b). The lead contamination originates from smelting activities at the Sandy City site and from historic mining operations at the Bingham Creek site. The lead concentrations in these soils, for the most part, appear to have a fairly homogenous and predictable distribution pattern. The Anaconda Smelter NPL Site is similar in that the lead in soil is distributed in a pattern which is consistent with the smelting activities and seems relatively homogenous within each of the gradients across the site. For this reason, it is reasonable to extrapolate the GSD from the site-specific data of the Sandy City Smelter Site and the Bingham Creek, Utah Site to the Anaconda Smelter NPL Site.

Because of the uncertainties associated with these default values, the results of the IEUBK model (as with any risk assessment) should not be viewed as exact. Actual risks could vary by a factor of 2 or 3.

4.0 TOXICITY ASSESSMENT

The purpose of the toxicity assessment is to review and summarize the potential for each COPC to cause adverse effects in exposed individuals. Adverse effects of chemical agents are generally dependent on the route of exposure, the duration and frequency of exposure, the concentration of chemical at the exposure point, and the sensitivity of people exposed.

Adverse health effects may follow acute (occurring soon after an exposure of a short duration), subchronic (occurring during or after an intermediate exposure period), or chronic (occurring during or after a long-term exposure) exposures to COPCs.

Of particular importance to many risk assessments is the potential for chemicals to cause cancer in exposed individuals. Cancer risks are assessed separately for chronic exposures to COPCs thought to have carcinogenic potential in humans.

For most adverse effects caused by chemicals, there is a positive relationship between dose and response. Generally, as dose increases, the type and severity of adverse responses also increases. For example, consumption of small amounts of alcohol, even on a daily basis, may have few effects other than slight euphoria. Consumption of larger amounts results in central nervous system depression (drunkenness) and even loss of consciousness. Consumed on a regular basis, larger amounts of alcohol can also cause severe disease such as liver cirrhosis. A key facet of any toxicity assessment is to use dose-response information to describe a quantitative relationship between human exposure and the potential for adverse health effects. For many chemicals, the assessment of dose-response has been completed (EPA 1995b). This report, therefore, includes only a summary of relevant dose-response information. References, which can provide detailed information, are cited at the end of this document.

Sources of toxicity information include, in order of priority, EPA's Integrated Risk Information System (IRIS) (EPA 1995b), Health Effects Assessment Summary Tables (HEAST) (EPA 1994d), EPA criteria documents, and Agency for Toxic Substances and

Disease Registry (ATSDR) Toxicological Profiles. This hierarchy of toxicological information sources is based on EPA guidance (EPA 1989a). However, only information from IRIS is used for toxicological evaluation of arsenic. Lead, which is the other COPC in the assessment, is unique in terms of risk assessment. Standard sources of toxicity information are not used for the evaluation of this metal. Lead risk assessment using the IEUBK Lead Model is discussed in more detail in Section 5.2.

Quantitative health risk assessment for most chemicals is based on the use of toxicological information to estimate toxicity criteria. These criteria are numerical expressions of the relationship between dose (exposure) and response (adverse health effects). As discussed below, separate criteria are developed for assessment of cancer and noncarcinogen health effects.

Toxicity criteria for carcinogens are provided as cancer slope factors (CSFs) in units of risk per milligram of chemical exposure per kilogram body weight per day. These factors are based on the assumption that no threshold for carcinogenic effects exists and any dose is associated with some finite cancer risk. Criteria for noncarcinogens, or for significant noncarcinogenic effects caused by carcinogens, are provided as reference doses (RfDs) in units of mg/kg-day. RfDs may be interpreted as thresholds below which adverse effects are not expected to occur even in the most sensitive populations.

Toxicity profiles are included for arsenic and lead, based in part on information in the documents cited above. These profiles outline major adverse effects, describe important toxicokinetic findings (absorption into, distribution in, metabolism by, and excretion from the body), discuss uncertainties and important data gaps, and summarize important studies used in the derivation of critical toxicity criteria.

Quantitative chemical dose-response information, in the form of critical toxicity criteria, is presented in Section 4.1. Uncertainties associated with toxicity criteria are discussed in

Section 4.2. Individual chemical profiles in support of toxicity criteria and a discussion of the uncertainty associated with the criteria are presented in Section 4.3.

4.1 TOXICITY CRITERIA

4.1.1 CARCINOGENS

Evidence of Carcinogenicity EPA has developed a system for stratifying evidence supporting classification of chemicals as carcinogens. This classification system characterizes the overall weight of evidence of carcinogenicity based on the availability of human, animal, and other supportive data (EPA 1989a). Three major factors are considered in characterizing weight of evidence of carcinogenicity: (1) the quality of evidence from human studies; (2) the quality of evidence from animal studies, and (3) other supportive data (e.g., studies of mutagenicity). The EPA classification system for the characterization of carcinogenicity has the following five categories:

- Group A – Human Carcinogen. This category indicates that there is sufficient evidence from human epidemiological studies to support a causal association between an agent and cancer.
- Group B – Probable Human Carcinogen. This category generally indicates that there is at least limited evidence from epidemiological studies of carcinogenicity to humans (Group B1) or that, in the absence of adequate data on humans, there is sufficient evidence of carcinogenicity in animals (Group B2).
- Group C – Possible Human Carcinogen. This category indicates that there is limited evidence of carcinogenicity in animals and no adequate data on humans.
- Group D – Not Classified. This category indicates that the evidence for carcinogenicity in both humans and animals is inadequate.

- Group E – Evidence of Noncarcinogenicity to Humans. This category indicates that there is evidence for noncarcinogenicity in at least two adequate animal tests in different species or in both epidemiological and animal studies.

Cancer Slope Factors The EPA Cancer Review and Validation Effort (CRAVE) has used a variety of specialized models to estimate the upper-bound risk of carcinogenesis for over 50 compounds. Data from animal or epidemiological studies are used to determine CSFs. A CSF relates the increase in an individual's risk of developing cancer over a 70-year lifetime to a unit of exposure (mg/kg-day). Units for CSFs are, thus, (mg/kg)/day)⁻¹.

When a CSF is multiplied by the lifetime average dose of a potential carcinogen (CDI), the product is the upper-bound lifetime individual cancer risk associated with exposure at that dose. This calculated risk is an estimate of the increased likelihood of cancer resulting from exposure to a COPC. For example, if the product of the CSF and the lifetime average daily dose is 1×10^{-6} , the predicted upper-bound excess cancer risk for the exposed population is one in one million (1:1,000,000). This risk would be in addition to any "background" risk of cancer not related to the chemical exposure. CSFs are provided in Table 4-1. Data used to develop these CSFs are found in the corresponding EPA health assessment and the open literature and are summarized in the toxicity profiles (Section 4.3).

The calculations of risk rely on the data derived from the results of human epidemiological studies or chronic animal bioassays. The likelihood that a chemical is a human carcinogen is a function of the weight of evidence of human and/or animal studies relating to:

- Increase in the number of tissues affected by the chemical
- Increase in the number of animal species, strains, sexes and number of experiments and doses showing a carcinogenic response
- Occurrence of clear dose-response relationships and a high level of statistical significance of the increased tumor incidence in treated compared to control groups

TABLE 4-1
 TOXICITY VALUES: POTENTIAL CARCINOGENIC EFFECTS
 ANA CONDA SMELTER SITE

Chemical	Oral Slope Factor	Inhalation Slope Factor	Weight of Evidence Classification	Type of Cancer	SF Basis/ SFSource	Date Online
Oral Route	(mg/kg-day) ⁻¹	(mg/kg-day) ⁻¹				
Arsenic	1.50E+00	1.50E+01	A	lung/skin	water; inhalation/IRIS	10/95
Lead	not available	not available	B2	NA	food/IRIS	3/1/88

NA = not applicable for B2 carcinogens

- A dose-related shortening of time-to-tumor occurrence or time-to-death with tumor
- A dose-related increase in the proportions of tumors that are malignant

Animal studies are usually conducted using relatively high doses in order to observe possible adverse effects. Since human exposures are generally expected to occur at lower doses, animal data are adjusted using mathematical models to predict cancer risk at low doses. Human epidemiologic data must often also be extrapolated to low doses using mathematical models, since human exposures information generally comes from studies of workers who have received much higher exposures than those expected following many environmental releases. Models used assume a linear dose response at low doses, and are generally assumed to provide conservative estimates of carcinogenic potential. These models, thus, provide only rough, but plausible, estimates of the upper limits on lifetime risk. Actual risks calculated using EPA slope factors are unlikely to be higher than those estimated, but they could be considerably lower, and may even be zero.

4.1.2 NONCARCINOGENS

RfDs are toxicity values developed by EPA for chemicals exhibiting noncarcinogenic effects after oral exposure. RfDs are usually derived from no-observable-adverse-effect levels (NOAELs) or lowest-observable-adverse-effect levels (LOAELs) taken either from human studies (often involving workplace exposures) or from animal studies.

Derivation of RfDs usually involves the use of uncertainty and modifying factors to extrapolate animal data to humans and/or to ensure the protection of sensitive human subpopulations. Uncertainty factors are applied, for example, to address the possibility that humans are more sensitive than experimental animals. In addition, uncertainty factors may be applied to account for sensitive subpopulations of humans, such as children, pregnant women,

and individuals with hay fever or asthma. Depending on the information available, modifying factors may also be applied. Such factors are sometimes applied, for example, to address lack of information on reproductive toxicity.

The RfD is an estimate of the daily exposure to a chemical that would be without adverse effects even if the exposure occurred continuously over a lifetime. An RfD is probably associated with an uncertainty spanning an order of magnitude or more. RfDs are presented in units of mg/kg-day for comparison with intake into the body. Intakes that are less than the RfD are not likely to be of concern. CDIs that are greater than the RfD indicate a possibility for adverse effects, at least in sensitive populations. However, whether such exposures actually produce adverse effects will (depending on the chemical) be a function of a number of factors such as the accuracy of uncertainty factors applied to the NOAEL or LOAEL, the appropriateness of animal models used in studies extrapolated to humans, and the potential for the chemical to cause effects in organs or systems (e.g., reproductive and immune systems) that have not been adequately studied. However, it is generally accepted that the protective assumptions made by EPA in deriving RfDs will, in most cases, mean there may be small risk of noncarcinogenic health effects for exposures slightly in excess of RfDs, with the probability of adverse effects increasing with increasing exposure. RfDs for noncarcinogenic effects are presented in Table 4-2.

4.2 UNCERTAINTIES ASSOCIATED WITH TOXICITY ASSESSMENT

There are many uncertainties associated with the use of toxicological information in health risk assessments that are related to uncertainties intrinsic to toxicology. Important among these are:

- The use of dose-response information from high-dose studies to predict adverse health effects at low doses

TABLE 4-2
 TOXICITY VALUES: POTENTIAL NONCARCINOGENIC EFFECTS
 ANACONDA SMELTER SITE

Chemical	Chronic RfD (mg/kg-day)	Confidence Level	Critical Effects	RfD Source/ RfD Basis	Uncertainty/ Modifying Factors	Date Online
Oral Route						
Arsenic	3.00E-04	medium	hyperpigmentation, etc.	epidemiology/IRIS	3	10/1/91
Lead	use IEUBK lead model		neurobehavioral development, etc			3/1/88

- The applicability of experimental animal studies to predict accurate health effects in humans
- The use of dose-response information from short-term exposure studies to predict adverse health effects of long-term exposures
- The use of toxicity values derived from homogenous animal populations or healthy human populations to predict adverse health effects in the general population, which is likely to contain sensitive individuals
- Quality of the study (i.e., design and conduct of the study)
- The selection criteria for the appropriate study used in the development of toxicity values

These and other uncertainties are limitations to the risk assessment process, which cannot be resolved quantitatively given the current understanding of toxicology and human health and using current risk assessment methodology. These uncertainties are addressed in part by consistent application of conservative assumptions regarding the toxic effects of chemicals, such as uncertainty factors for RfDs and upper bound estimates for CSFs. Such procedures are intended to protect public health in the absence of data and may, in many cases, overstate potential impacts on human health.

4.3 TOXICITY PROFILES

4.3.1 ARSENIC

Arsenic (As) is a naturally occurring element found in a variety of complex sulfidic ores. Arsenic trioxide, produced primarily from flue dust that is generated at copper and lead smelters, is the most important commercial arsenic compound. Production of arsenic trioxide in the United States ceased in 1985. Since then, importation of elemental arsenic and arsenic trioxide has increased dramatically. Arsenic is used in wood preservatives and in agricultural insecticides and herbicides.

Toxicokinetics

Absorption of arsenic from the gastrointestinal tract is dependent on the solubility of the arsenic compound. Soluble forms of both As(III) and As(V) are almost completely absorbed in laboratory animals (Vahter 1983, Battelle 1994) and humans (EPA 1984a, b). Bettley and O'Shea (1975, in ATSDR 1991a) reported that 5% of arsenite was recovered in the feces of humans orally exposed to arsenite. Insoluble forms may not be available for absorption in humans as indicated by the lack of increase in urinary excretion of arsenic in human volunteers administered arsenic selenide orally (Mappes 1977).

Site-specific estimates of arsenic absorption from ingested soil were developed by EPA for this HHRA (EPA 1994a, 1995a). Data used to develop site-specific arsenic absorption estimates describe the arsenic concentrations in blood, urine, and feces collected from Cynomologus monkeys exposed to arsenic by intravenous injection, gavage, and oral administration of capsules containing soil or dust collected in Community soils OU (Battelle 1994). Mean absolute absorption estimates derived from urine arsenic estimates were 91%, 18.3%, and 25.8% for gavage, soil, and dust, respectively. Absolute absorption estimates derived from blood arsenic concentrations were similar and ranged between 91% and 100% for gavage, 11% and 18% for soil ingestion, and 8% and 11% for dust ingestion. Discussion of the derivation of these values is provided in Appendix C.

Following inhalation, absorption of arsenic is dependent on particle size, with larger particles being quickly cleared from the lungs with little absorption. In one study, Holland *et al.* (1959, in ATSDR 1991a) examined the absorption and deposition of arsenic in lung cancer patients exposed to arsenic in arsenite-containing cigarette smoke and arsenic-containing aerosols. In the patients, approximately 40% of arsenic particulates were deposited in the lungs and approximately 75-85% of the deposited arsenic was absorbed by the lungs. Smaller particles penetrate into alveolar spaces and may remain there for extended periods, increasing the chances for inhaled arsenic to be absorbed (EPA 1984a, b). Absorption from the lung

may be rapid for soluble arsenic forms, but is much slower for more insoluble forms (ATSDR 1991a).

No studies are available regarding the absorption of arsenic in humans following dermal exposure. Animal studies indicate that arsenic may bind to the skin following dermal exposure, and be slowly absorbed even after exposure ends (ATSDR, 1991a). In one study in which the tails of rats were immersed in sodium arsenate for 1 hour, arsenic uptake was not detected for up to 24 hours after exposure; however, over the next five days arsenic concentrations rose in the blood, liver and spleen. The rate of uptake was estimated to be 1 to 33 micrograms per squared centimeter per hour ($\mu\text{g}/\text{cm}^2/\text{hr}$) (ATSDR 1991a).

Following absorption, arsenic is distributed throughout the body. Analysis of autopsy tissues collected from humans exposed to background levels of arsenic in food show that arsenic was present in all tissues of the body (ATSDR 1991a). Similarly, elevated levels of arsenic were noted in all tissues of mice and hamsters given oral doses of arsenate or arsenite (ATSDR 1991a). Rhoads and Sanders (1985, in ATSDR 1991a) reported that distribution of arsenic trioxide after intratracheal administration to rats was to the liver, kidneys, skeleton, gastrointestinal tract, and other tissues. No organ appears to preferentially accumulate arsenic.

Metabolism of inorganic arsenic takes place via two major processes: (1) oxidation/reduction reactions that interconvert arsenate and arsenite, and (2) methylation reactions which convert arsenite to monomethyl arsenic acid (MMA) and dimethyl arsenic acid (DMA). These processes appear to be used for metabolism regardless of the route of exposure.

Arsenic is efficiently metabolized to methylated forms in the liver in both animals (ATSDR 1991a) and humans (Buchet, *et al.* 1981). Because acute toxicity of these methylated forms is much less than for inorganic arsenic, methylation is considered detoxification. At high arsenic doses, methylation pathways may become saturated (Buchet, *et al.* 1981). This may

result in a "threshold" determined by the ability to metabolize arsenic, where low doses are relatively nontoxic due to conversion to methylated forms, and higher doses are more toxic since greater amounts of inorganic arsenic will be available for distribution to target tissues. This is especially important for carcinogenesis following oral exposure, where small daily intakes could be much less effective in inducing cancer than higher doses that saturate metabolism. Unfortunately, available information is insufficient to determine the saturation point in humans (EPA 1988b) and it is not possible at this time to make adjustments to the oral CSF for low CDIs.

Most arsenic is promptly excreted in the urine in the form of metabolic products, including As(+3), As(+5), DMA, and MMA (ATSDR 1991a). Vahter *et al.* (1986, in ATSDR 1991a) reported that urinary arsenic levels in smelter workers rose within hours of starting work on a Monday and then fell over the weekend. This indicates that excretion is rapid, an observation supported by experimental studies in animals (Rhoads and Sanders 1985, Marafante and Vahter 1987, both in ATSDR 1991a). Human oral exposure to known amounts of arsenite or arsenate indicate that very little is excreted in the feces (Bettley and O'Shea 1975, in ATSDR 1991a), while 45-85% is excreted in the urine between 1-3 days (Buchet *et al.* 1981, Crecelius 1977, Mappes 1977, Tam *et al.* 1979, all in ATSDR 1991a). Small amounts of arsenic may remain bound to tissues, depending inversely on the rate and extent of methylation (ATSDR 1991a).

Qualitative Description of Health Effects

Acute exposure to ingested arsenic may result in death (ATSDR 1991a). Although the information on lethal doses to humans is sparse, Armstrong *et al.* (1984, in ATSDR 1991a) reported that two people in a family of eight died after ingesting 110 ppm of arsenic in water. A number of serious cardiovascular effects may result after acute and chronic ingestion. These effects include myocardial depolarization, cardiac arrhythmias, and damage to the vascular system (Glazener *et al.* 1968, Goldsmith and From 1986, Heyman *et al.* 1956, Little

et al. 1990, Mizuta *et al.* 1956, Tseng 1977, all in ATSDR 1991a). An example of vascular damage is "blackfoot disease," a disease characterized by loss of circulation in hands and feet which leads to necrosis and gangrene (Chen *et al.* 1988, Chi and Blackwill 1968, Tseng 1977 1989, Tseng *et al.* 1968, all in ATSDR 1991a). The disease was endemic in an area of Taiwan where the population was exposed to arsenic ranging from 0.17 to 0.8 ppm in well water. Oral ingestion of arsenic can affect the skin. Hyperkeratosis, hyperpigmentation, and hypopigmentation have been observed on the faces, necks, and backs of workers following chronic oral exposure (ATSDR 1991a). Studies indicate that arsenic may be fetotoxic, teratogenic, and embryotoxic in animal tests (ATSDR 1991a).

There is convincing evidence from a large number of studies that ingestion of arsenic increases the risk of skin cancer. EPA (1995b) has classified arsenic via oral exposure in Group A – Human Carcinogen. Squamous cell carcinomas are the most common types of skin cancer and appear to develop from hyperkeratinized corns. Basal cell carcinomas also occur. In a key study by Tseng *et al.* (1968, in ATSDR 1991a), ingestion of contaminated drinking water from wells in Taiwan was correlated with an increased skin cancer rate. Based on an examination of over 40,000 people in Taiwan, the skin cancer rate was 10.6/1,000. There is also mounting evidence that ingestion of arsenic may increase the risks of internal cancers. These include tumors of the bladder, kidney, liver, and lung (ATSDR 1991a, EPA 1995b).

After acute exposure to arsenic compounds via inhalation, humans may experience irritation of the mucous membranes in the nose and throat, which may lead to laryngitis, bronchitis, rhinitis, and in very high doses, perforation of the nasal septum (Dunlap 1921, Morton and Caron 1989, Pinto and McGill 1953, all in ATSDR 1991a). Chronic inhalation of arsenic compounds may lead to an increased risk of mortality from cardiovascular disease (Axelson *et al.* 1978, Lee-Feldstein 1983, Wall 1980, all in ATSDR 1991a), but this effect has not been observed in all studies. An increased incidence of Raynaud's disease (cyanosis of the digits due to arterial and arteriolar contraction) and increased constriction of blood vessels in

response to cold, suggests that long-term inhalation exposure to arsenic compounds (0.05-0.5 mg As/m³) may injure blood vessels and/or the heart (Lagerkvist *et al.* 1986, Lagerkvist *et al.* 1988, both in ATSDR 1991a).

There is convincing evidence that chronic arsenic inhalation exposure increases the risk of lung cancer. EPA (1995b) has classified arsenic according to its weight of evidence criteria in Group A – Human Carcinogen (via inhalation). Most studies involved workers in copper smelters exposed to arsenic trioxide in the air, but an increased risk of lung cancer has also been observed at chemical plants where workers were exposed to arsenate. Several studies also suggest that residents living near smelters or arsenic chemical plants may also have an increased risk of lung cancer, although an increased cancer risk was not observed in all cases (ATSDR 1991a, EPA 1995b).

Quantitative Description of Health Effects

EPA derived an oral RfD based on a study by Tseng *et al.* (1968; in ATSDR 1991a) and Tseng (1977, in ATSDR 1991a). Tseng *et al.* (1968, in ATSDR 1991a) and a follow-up study by Tseng (1977, in ATSDR 1991a) observed a population in Taiwan where well water was contaminated with arsenic. The Tseng (1977, in ATSDR 1991a) study reported an increased incidence of blackfoot disease that was both age- and dose-specific. For the low-dose group (170 µg/L), blackfoot disease was observed in 4.6 per 1,000 for the 20–39-years-of-exposure group, 10.5 per 1,000 for the 40–59-years-of-exposure group, and 20.3 for the "greater-than-60-years-of-exposure" group. In addition, an increased incidence of hyperpigmentation and keratosis occurred with increasing age (Tseng *et al.* 1968, in ATSDR 1991a). Based on effects of hyperpigmentation, keratosis, and blackfoot disease, the LOAEL was 0.014 (mg/kg)/day = ((170 µg/L x 4.5 L/day) + 2 µg/day [contribution of food]). The NOAEL was 0.0008 (mg/kg)/day = ((9 µg/L x 4.5 L/day) + 2 µg/day). This estimate is based on the arithmetic mean concentration of arsenic in the well used by the individuals in the control group (9 µg/L, range 1-17 µg/L) (Abernathy *et al.* 1989, in EPA 1995b). To

derive an oral RfD, an uncertainty factor of 3 was applied to the NOAEL to account for the lack of reproductive toxicity data and to account for sensitive individuals. Thus, the oral RfD is 3×10^{-4} mg/kg-day (EPA 1995b).

The studies on which the RfD are based have been given a medium level of confidence, based on the presence of other contaminants and poor characterization of the exposure doses. The supporting human toxicity database is extensive but lacking in some important areas. However, it does support the choice of NOAEL and is given a medium degree of confidence. Therefore, medium confidence is placed in the oral RfD (EPA 1995b).

Tseng (1977, in ATSDR 1991a) observed a population in Taiwan where well water contaminated with arsenic was used for 60 years. The study found significantly elevated standard mortality ratios for cancer of the bladder, lung, liver, kidney, skin, and colon. The study was extensive, but did not define a control population. Concentrations of arsenic in the water ranged from 0.01 to 1.82 mg/L. The overall prevalence rate for skin cancer was 10.6 per 1,000 and for peripheral vascular disorder of the extremities was 8.9 per 1,000. Three dose groups were designated as "low" (below 0.3 mg/L), "mid" (0.3-0.6 mg/L), and "high" (above 0.6 mg/L). Tseng (1977, in ATSDR 1991a) reported a dose-response relationship between concentrations of arsenic in the water and skin cancer. Based on this study, the oral CSF is $1.50 ((\text{mg/kg})/\text{day})^{-1}$ (EPA 1995b).

EPA has derived an inhalation CSF of $15 ((\text{mg/kg-day})^{-1})$ based on six occupational exposure studies of two different exposed populations (Brown and Chu 1983a, Brown and Chu 1983b, Brown and Chu 1983c, Lee-Feldstein 1983, Higgins *et al.* 1982, Enterline and Marsh 1982, all in ATSDR 1991a). These studies have reported an association between occupational exposure to arsenic and lung cancer mortality. To derive the inhalation CSF, the geometric mean was taken within each of the exposed populations and the final inhalation CSF was the geometric mean of the two exposed populations. Supporting evidence of the carcinogenicity

of arsenic has also been found in residents drinking arsenic-containing water and residents living near a pesticide manufacturing plant (EPA 1995b).

The described CSFs are based on the absorbed dose in contrast to those for most other chemicals which are based on administered doses. EPA assumed 100% absorption of arsenic following oral exposure from water and 30% absorption following inhalation exposure (EPA 1988b; EPA 1995b).

The maximum contaminant level goal (MCLG) of 0.05 mg/L has been proposed, based on the current maximum contaminant level (MCL) of 0.05 mg/L. Although arsenic is potentially carcinogenic, its potential essential nutrient value was considered in determining the MCLG (EPA 1995b).

SUMMARY OF ARSENIC CRITERIA

		SOURCE
EPA Carcinogenic Classification	Group A	EPA 1995b
Maximum Contaminant Level (MCL)	0.05 mg/L	EPA 1993b
Chronic Oral RfD	3.0E-04 (mg/kg)/day	EPA 1995b
Oral SF	1.5E+007 ((mg/kg)/day) ⁻¹	EPA 1995b
Inhalation SF	1.5E+01 ((mg/kg)/day) ⁻¹	EPA 1995b
EPA Drinking Water Health Advisory (HA)	Not Available	EPA 1993b
Ambient Water Quality Criteria (AWQC)		
Human – Water and Fish Consumption	2.2E-03 µg/L	EPA 1995b
Aquatic Organisms		
Freshwater		
Acute	3.6E+02 µg/L	EPA 1995b
Chronic	1.9E+02 µg/L	EPA 1995b
Marine		
Acute	6.9E+01 µg/L	EPA 1995b
Chronic	3.6E+01 µg/L	EPA 1995b

4.3.2 LEAD (INORGANIC)

Lead (Pb) is a naturally occurring, ubiquitous metal. Concentrations in rocks and soils in the western United States range from 10 to 700 mg/kg (Shacklette and Boerngen 1984). Lead is often found in association with cadmium, zinc, and silver ores. Lead is obtained through

underground mining, as a by-product from open pit copper mines, and from secondary sources including scrap, product wastes, refinery drosses, and residues. During 1989, United States mine production of recoverable lead was 905 million pounds and production of refined lead from primary sources was 874 million pounds. Lead production by recovery from secondary sources was 1.783 billion pounds. Lead imports have been decreasing: in 1977, 719 million pounds were imported, while in 1989, 281 million pounds of lead were imported into the United States. Lead is commercially important because it is very soft, highly malleable, ductile, and a poor conductor. In addition, it is resistant to corrosion, is an effective sound absorber, and makes an excellent radiation shield. Historically, major uses of lead included battery casings, pigments in paint, solders, and as gasoline additives. Because of its extensive use and its ubiquitous distribution, exposure to lead is common.

Toxicokinetics

Oral absorption of inorganic lead in humans ranges from as low as 3% to as high as 80% (ATSDR 1991b). The percentage of absorbed lead appears to be dependent on the solubility of the lead salt ingested as well as age, nutritional status, and fasting time. Dietary absorption of lead has been reported as 50% and 15% for children and adults, respectively (Chamberlain *et al.* 1978; in ATSDR 1991b). Animal studies indicate that particle size also influences lead absorption from the gastrointestinal tract (EPA 1986a in ATSDR 1991). For example, in rats an inverse relationship was shown between lead absorption for metallic lead. Tissue concentrations of lead increased by 2.3-fold when particle size of small lead particles less than (<) 38 micrometers (μm) were administered as compared to lead particles measuring 150 to 250 μm (ATSDR 1991b). As described in Section 3.3.2.3, this HHRA uses a default value of 30% for lead absorption from soil and dust ingestion (EPA 1991e).

Absorption of inhaled lead is thought to reach 100%; however, not all inhaled particles are deposited in the respiratory tract. The deposition rate of lead-containing particles is

influenced by factors such as particle size and ventilation rate, and is estimated to be between 30% and 50% of the inhaled particles.

Dermal absorption of lead is not considered a significant pathway. Route of absorption does not effect distribution of lead. After absorption, lead is distributed among several physiologically distinct compartments, including blood, soft tissue, particularly brain, kidney and liver, and bone (ATSDR 1991b). In adults, approximately 94% of the total body burden is in bone (ATSDR 1991b). Estimates of elimination half-times for lead from blood range from 15 to 35 days and elimination half times from other soft tissues are probably similar (Harley and Kneip 1985; in ATSDR 1991b). Elimination half-times for lead from mineralized bone are expressed in years. Because metabolic stress such as pregnancy may result in increased bone turnover or demineralization, there is potential for a portion of the parental bone lead-burden to be transferred to the fetus.

Inorganic lead is not metabolized or biotransformed in the body (ATSDR 1991b). All absorbed lead that is not retained is excreted by the kidney or through biliary clearance into the gastrointestinal tract. Infants (0 to 2 years of age) retain approximately 32% of the lead absorbed (Ziegler *et al.* 1978; in ATSDR 1991b) whereas adults retain only about 1% of absorbed lead (Rabinowitz *et al.* 1977; in ATSDR 1991b). Most toxicity endpoints associated with exposure to lead can be correlated with blood-lead levels. Blood-lead levels are, therefore, a useful index of toxicity.

Qualitative Description of Health Effects

Cases of severe lead encephalopathy have resulted in death in both adults and children. Blood-lead levels associated with death in children have ranged from approximately 125 micrograms of lead per deciliter blood ($\mu\text{g Pb/dL}$) to 750 $\mu\text{g Pb/dL}$. Lead encephalopathy (non-fatal) has been seen at blood-lead levels of 60-300 $\mu\text{g/dL}$. At lower blood-lead levels, systemic effects associated with lead intoxication include increased systolic and diastolic

blood pressure (Harlan 1988; Pocock *et al.* 1984, 1985; in ATSDR 1991b). Harlan's analysis, which is based on National Health and Nutrition Examination Survey (NHANES II) data, estimated an increase in blood pressure of 7 mm Hg at blood-lead levels between 14 and 30 µg Pb/dL. Pirkle *et al.* (1985; in ATSDR 1991b) evaluated the same data set for 40-59-year-old white males and found no threshold for increased blood pressure associated with increased blood-lead levels across the range of 7-34 µg Pb/dL. Gastrointestinal symptoms such as colic, abdominal pain, constipation and anorexia are typically seen at blood-lead levels of 100-200 µg Pb/dL but have been reported at blood-lead levels as low as 40 µg Pb/dL.

Lead is known to depress heme synthesis and this effect appears to have no threshold in the range of available blood-lead concentration data. Cytochrome P450 formation is also inhibited in the presence of lead. Kidney damage occurs with both acute and chronic exposures to lead. Acute renal toxicity has been reported in lead-intoxicated children and is considered reversible, whereas chronic renal toxicity has been observed in lead-exposed workers and is considered irreversible. Lead interferes with vitamin D metabolism and may have some effect on the cellular component of the immune system.

The lowest-observed-effect-level (LOEL) for overt neurotoxic toxicity in adults is estimated to be 40 µg Pb/dL (ATSDR 1991b). Early symptoms include irritability, poor attention span, headache, muscular tremor, loss of memory and hallucinations. As the condition worsens, symptoms include delirium, convulsions, paralysis and coma and may lead to death. Decreased peripheral nerve conduction velocities (NCVs) have been seen in workers at blood-lead levels ranging from 30-48µg Pb/dL; these effects are probably reversible.

Neurotoxicity in children is seen at very low blood-lead levels. Low-level prenatal exposure to lead has been shown to result in reduced birth weight and gestation age, as well as neurobehavioral deficits or delays (ATSDR 1991b). Prenatal exposure was generally estimated through maternal or cord blood-lead concentrations. Postnatal lead exposures may

result in fine motor dysfunction, hyperactivity, and altered behavioral patterns (ATSDR 1991b). Several studies have demonstrated a statistically significant decrement in children's intelligence quotients (IQs) when correlated with blood-lead levels. Subtle signs of lead-induced effects begin to be apparent at blood-lead levels of 10 µg/dL or even lower, with effects becoming clearer by 30 to 40 µg/dL. Some researchers claim that some of the effects of lead, including neurobehavioral effects, heme synthesis depression, and fetal developmental problems, do not have a threshold value (EPA 1994c).

Studies on association of occupational exposure to lead with increased cancer risks are insufficient to determine the carcinogenicity of lead in humans. Ingestion of lead acetate and lead phosphate produced renal tumors in laboratory rats and mice.

The literature on lead is difficult to summarize briefly. The Toxicological Profile for Lead (ATSDR 1991b) contains over 1,000 references, and much of the brief synopsis above is taken from the profile.

Quantitative Description of Health Effects

EPA has not published a RfD or acceptable intakes for chronic or subchronic periods of human exposure in IRIS (EPA 1995b) or HEAST (EPA 1994d) because available data suggest no threshold for adverse effects even at exposure levels that might be considered background. Any significant increase above such exposures could represent a cause for concern. In lieu of acceptable intake for chronic exposure (AIC) or RfDs, EPA has developed a biokinetic computer model for prediction of blood-lead levels in children exposed to lead from a variety of sources, including soil, dust, air, diet, lead-based paint, and maternal blood. Estimated blood-lead levels are compared to target blood-lead concentrations to assess possible risks.

The model can be used to assess risks to individual children or a population of children. For a single child, risk is calculated as the probability that the child's blood-lead level will exceed

the level of concern (10 µg/dL) (EPA 1991c). The single-child assessment is generally used to evaluate remedial options on a house-by-house or yard-by-yard basis (EPA 1991e). For a population of children, risk is expressed as the percentage of children that are likely to have a blood lead level greater than 10 µg/dL. This HHRA evaluates lead risks to populations of children. Protection of young children is considered achieved when model results indicate that less than 5% of the population of children will have blood-lead levels greater than 10 µg/dL (EPA 1994c). Because children between the ages of 0-6 are thought to be most susceptible to the adverse effects of lead, protection for this age group (0-6 years old) is assumed to also protect older individuals.

No inhalation reference concentration (RfC) is available for lead, and, as discussed above, it is not clear that there is a practical threshold below which there are no risks from exposure to lead. Since RfCs are based on the assumption that such a threshold exists, the estimation of an RfC for lead is not appropriate.

Oral ingestion of certain lead salts (lead acetate, lead phosphate, lead subacetate) have been associated with increased renal tumor frequency in rats (Azar *et al.* 1973; Koller *et al.* 1985; both in ATSDR 1991b), but no quantitative estimate of excess cancer risk has been developed by CRAVE. EPA (1987, in EPA 1995b) has noted that the available data provide an insufficient basis on which to regulate lead acetate, lead phosphate, and lead subacetate as human carcinogens. However, applying the criteria described in EPA's Guidelines for Carcinogenic Risk Assessment (EPA 1986a), these lead salts have been classified by EPA (1987; in EPA 1995b) in Group B2 – probable human carcinogen.

At present, standards for lead in soil have not been established in the United States. The United Kingdom Directorate of the Environment has developed a tentative guideline of 550 ppm for lead in soil in residential areas (Smith *et al.* 1981, in ATSDR 1991b). Vernon Houk of the Centers for Disease Control has been quoted as indicating that levels of lead in soil of 300-400 ppm are acceptable based on studies of childhood lead poisoning (Mielke *et al.* 1984,

in ATSDR 1991b). Recently, EPA has proposed a screening level of 400 ppm for lead in residential soils (EPA 1994c).

The current MCL for lead (at source) is 0.05 mg/L, as stated in the Code of Federal Regulations at 40 CFR 141.11. The Treatment Technique Action Level of 0.015 mg/L has been finalized (EPA 1991d, in ATSDR 1991b) by the Office of Drinking Water. The MCLG for lead at the source and at the tap is zero.

The EPA Office of Drinking Water issued a draft health advisory of 20 µg/day for all extended periods of exposure (EPA 1985, in ATSDR 1991b). Blood-lead levels above 10 µg/dL are identified as of concern, and children under six years of age are assumed to be the most sensitive subpopulation (CDC 1991).

The National Primary and Secondary Ambient Air Quality Standard for lead is 1.5 µg/m³. This standard is currently being re-evaluated (40 CFR 50.12).

SUMMARY OF LEAD CRITERIA

SOURCE

EPA Carcinogenic Classification	Group B2	EPA 1995b
Maximum Contaminant Level (MCL)	0.05 mg/L	40 CFR 141.11
Maximum Contaminant Level Goal (MCLG)	0 mg/L	40 CFR 141.11
Treatment Technique Action Level	0.015 mg/L	EPA 1995b
EPA Drinking Water Health Advisory (HA)	NA	
Ambient Water Quality Criteria (AWQC)		
Human Water and Fish Consumption	0.05 mg/L	EPA 1992c
Aquatic Organisms		
Freshwater		
Acute	8.2E+1 µg/L	EPA 1992c
Chronic	3.2E+0 µg/L	EPA 1992c
Marine		
Acute	2.2E+2 µg/L	EPA 1992c
Chronic	8.5E+0 µg/L	EPA 1992c

5.0 RISK CHARACTERIZATION

In this section, potential site-related risks are characterized for exposure to arsenic and lead for exposure pathways selected in Section 3.2. In Section 5.1, toxicity values for arsenic (Section 4.0) are combined with estimates of CDI (Section 3.3.3) to calculate cancer and noncarcinogenic health risks for each exposure pathway. Total risks, assuming exposure from each pathway, are also discussed. Risks associated with exposure to lead are assessed in Section 5.2 using the IEUBK Lead Model, Version 0.99. Children (aged 0-6) are considered the sensitive subpopulation at risk for adverse effects due to exposure to lead in environmental media. Risks to adults from lead exposure are not evaluated. Uncertainties in the risk assessment are presented in Section 5.3. A summary of risk estimates and associated uncertainties is provided in Section 5.4.

5.1 ARSENIC HEALTH RISKS

Arsenic is known to cause both systemic toxicity and cancer following prolonged exposure. Therefore, both cancer and noncancer health risks are assessed.

5.1.1 CANCER HEALTH RISKS FROM ARSENIC EXPOSURE

To evaluate cancer risks due to exposure to arsenic, pathway-specific CDI estimated previously are multiplied by the arsenic-specific oral CSF. Since inhalation exposures are not considered significant, the inhalation slope factor is not used in the assessment.

Table 5-1 presents pathway-specific and total cancer risks for RME and CTE scenarios. Potential risks based on RME estimates associated with ingestion of soil/interior dust are in the range of 2×10^{-5} to 4×10^{-5} for all subareas, reflecting the relatively homogeneous distribution of arsenic in the study area. The highest risks are estimated for subareas D and F1, perhaps reflecting the proximity of these areas to Smelter Hill. However, differences in

TABLE 5-1
CANCER RISKS
INGESTION OF ARSENIC IN GROUNDWATER, SOIL, AND DUST
RME AND CTE RESIDENTIAL SCENARIO
ANACONDA SMELTER SITE
(mg/kg-day)

SUBAREA	RME SCENARIO			CTE SCENARIO		
	Groundwater	Soil and Dust	Total	Groundwater	Soil and Dust	Total
	Ingestion	Ingestion	Arsenic	Ingestion	Ingestion	Arsenic
	Cancer Risk	Cancer Risk	Cancer Risk	Cancer Risk	Cancer Risk	Cancer Risk
Subarea A	3.76E-05	1.55E-05	5.30E-05	3.94E-06	2.44E-06	6.38E-06
Subarea B	0	2.05E-05	2.05E-05	0	3.23E-06	3.23E-06
Subarea C	0	2.64E-05	2.64E-05	0	4.17E-06	4.17E-06
Subarea D	0	4.18E-05	4.18E-05	0	6.59E-06	6.59E-06
Subarea E	0	2.79E-05	2.79E-05	0	4.40E-06	4.40E-06
Subarea F1	0	3.84E-05	3.84E-05	0	6.06E-06	6.06E-06
Subarea F2	0	3.29E-05	3.29E-05	0	5.19E-06	5.19E-06
Subarea I	0	2.53E-05	2.53E-05	0	3.98E-06	3.98E-06
Subarea J	0	2.43E-05	2.43E-05	0	3.83E-06	3.83E-06
Opportunity	3.16E-05	2.34E-05	5.51E-05	3.32E-06	3.69E-06	7.01E-06

risk estimates among subareas are small and may not be significant. Risks based on CTE estimates are about 16% of those based on RME.

Potential risks from ingestion of arsenic in groundwater are somewhat higher than those for soil/dust ingestion in subarea A and in Opportunity, although they still fall within the EPA risk range. Groundwater risks are not evaluated for other subareas since data from these areas are lacking. All estimated risks fall within the risk range of 1×10^{-4} and 1×10^{-6} , which are generally considered acceptable by EPA (1991b).

5.1.2 NONCARCINOGENIC HEALTH RISKS FROM ARSENIC EXPOSURE

To evaluate noncarcinogenic health risks, CDI (Section 3.3.3) are compared to toxicity criteria to determine if exposure might exceed the nominal threshold established by the RfD. The ratio of exposure estimate to toxicity criteria for a single chemical is called a hazard quotient (HQ) and provides a measure of risk for systemic health effects. The ratio is calculated as:

$$\text{Hazard quotient} = \frac{\text{CDI}}{\text{RfD}}$$

Where: CDI = Chronic Daily Intake ((mg/kg)/day)
RfD = Reference Dose ((mg/kg)/day)

The HQ assumes that there is a level of exposure (the RfD) below which it is unlikely for even a sensitive population to experience adverse health effects. If the CDI exceeds the RfD, a potential for non-cancer adverse health effects may exist. Although a quantitative estimate of risk cannot be established for noncancer effects, it is assumed that a small exceedance of the RfD might be associated with a small risk and that risks would increase with larger CDIs.

At most Superfund Sites, one must assess potential health effects of more than one chemical. The Hazard Index (HI) is used to assess the overall potential for systemic effects posed by exposure to multiple chemicals, and is equal to the sum of HQs for all of the COPCs for a

group of receptors. When the HI exceeds one, there may be a potential for adverse health effects from exposure to all of the chemicals at the site, and further evaluation of mechanisms of toxic action is required. For this HHRA, however, HQs are calculated only for arsenic. As described in Section 4.0, lead risks are evaluated through the use of the EPA IEUBK Lead Model, Version 0.99. This model evaluates health risks based on blood-lead levels. It would be inappropriate to attempt to combine arsenic and lead toxicity values because of the different evaluation methodologies. Additionally, lead and arsenic do not induce similar toxic effects, nor does their toxicity occur through the same mechanism of action. Therefore, the HI approach is not applicable to this HHRA.

Table 5-2 presents noncarcinogenic HQs for arsenic for each subarea. Based on RME estimates, HQs for ingestion of soil and dust range from about 0.1 to 0.3 for all subareas. As with cancer risk estimates, the highest HQs are found in subareas D and F1, though differences among subareas are small. HQs based on CTE estimates are about 53% of those based on RME.

Potential risks due to ingestion of groundwater are similar to those for ingestion of soil/dust in subarea A and in Opportunity, and overall these risks fall in the lower half of the range of HQs for soil/dust ingestion. The highest HQ (0.34 for subarea A) is less than one suggesting that exposures to arsenic in groundwater will not exceed the target HQ of 1.

All HQs estimated are less than unity, suggesting little potential for impacts to human health. Thus, neither cancer risks nor hazard quotients exceed common criteria used to establish acceptable risk levels. Considering these criteria alone, potential arsenic exposure in the communities of Anaconda and Opportunity does not appear to be associated with unacceptable health risks.

TABLE 5-2

NONCANCER RISKS
 INGESTION OF ARSENIC IN GROUNDWATER, SOIL, AND DUST
 RME AND CTE RESIDENTIAL SCENARIO
 ANACONDA SMELTER SITE
 (mg/kg-day)

SUBAREA	RME SCENARIO			CTE SCENARIO		
	Groundwater	Soil and Dust	Total	Groundwater	Soil and Dust	Total
	Ingestion	Ingestion	Arsenic	Ingestion	Ingestion	Arsenic
	HQ	HQ	Risk	HQ	HQ	Risk
Subarea A	3.37E-01	2.11E-01	5.48E-01	1.34E-01	1.13E-01	2.46E-01
Subarea B	0	2.79E-01	2.79E-01	0	1.49E-01	1.49E-01
Subarea C	0	3.60E-01	3.60E-01	0	1.93E-01	1.93E-01
Subarea D	0	5.70E-01	5.70E-01	0	3.05E-01	3.05E-01
Subarea E	0	3.80E-01	3.80E-01	0	2.03E-01	2.03E-01
Subarea F1	0	5.24E-01	5.24E-01	0	2.80E-01	2.80E-01
Subarea F2	0	4.48E-01	4.48E-01	0	2.40E-01	2.40E-01
Subarea I	0	3.45E-01	3.45E-01	0	1.84E-01	1.84E-01
Subarea J	0	3.32E-01	3.32E-01	0	1.77E-01	1.77E-01
Opportunity	2.83E-01	3.20E-01	6.03E-01	1.12E-01	1.71E-01	2.83E-01

5.1.3 COMBINED RISKS

Residents of Anaconda and Opportunity might be exposed to both contaminated soil/dust and to contaminated groundwater. Thus, total risks for receptor populations may be higher than risks estimated for individual pathways. It may be appropriate to combine risks based on RME estimates if it is likely that the same individual might experience RME exposures in more than one pathway. For Anaconda and Opportunity, it is conceivable that the same individuals could be exposed at high levels to both soil/dust and groundwater. In fact, within a single subarea, soil concentrations are relatively consistent, suggesting that the occurrence of high soil/dust levels and high local groundwater contamination in the same location is likely. Though this alone does not indicate that people at such location will be maximally exposed to both soil/dust and groundwater, it does increase the likelihood for co-occurrence of such exposures. Thus, it seems reasonable to combine risks based on RME for subarea A and Opportunity.

Combined RME cancer risks for subarea A (5.3×10^{-5}) and Opportunity (5.5×10^{-5}) are still within the EPA acceptable risk range (Table 5-1). Likewise, combined HQs (0.55 and 0.6 for subarea A and Opportunity respectively) are still below the target HQ of one (Table 5-2). Thus, combining risks from soil/dust ingestion and groundwater ingestion pathways does not result in risk estimates that exceed common criteria for acceptable risk.

5.2 POTENTIAL HEALTH RISKS ASSOCIATED WITH EXPOSURE TO LEAD

Risks from exposure to lead cannot be assessed using standard methods, because toxicological criteria for lead are not available. The EPA's position is that current data are insufficient to determine an RfD or RfC for lead. Further, EPA feels that the primary threat to human health from exposure to lead is subtle neurological effects in young children. For this reason, the EPA has not derived a CSF for lead, despite the chemical's Group B2 status as a probable human carcinogen.

The best available quantitative tool for evaluating health effects from exposure to lead is the IEUBK model (EPA 1994b). This model uses current information on the uptake of lead following exposure from different routes, its distribution among various internal body compartments, and its excretion, to predict impacts of lead exposure on blood-lead concentrations in young children. Predicted blood-lead concentration can then be compared with target blood-lead concentrations associated with subtle neurological effects in children. Because children are thought to be most susceptible to the adverse effects of lead, protection for this age group is assumed to also protect older individuals. Protection of young children is considered achieved when the model predicts that less than 5% of children will have blood-lead levels greater than 10 µg/dL (EPA 1994c).

The IEUBK Lead Model, Version 0.99, is used to evaluate potential risks from exposure to lead associated with the Anaconda Smelter NPL Site. Young children (0 to 6 years old) who live near the Anaconda Smelter NPL Site are evaluated for potential exposure to lead in soil and dust.

IEUBK Modeling Results

Results from IEUBK modeling runs are presented in Appendix E to this document. The probability plots for blood-lead concentrations for exposure subareas in Anaconda and Opportunity are presented on Figures 5-1 to 5-11. Table 5-3 summarizes the modeling results. Modeling predicted that 5% of children in exposure subarea E may have blood-lead levels in excess of 10 µg/dL. The estimated percentage of individuals in exposure subarea E having blood-lead levels above 10 µg/dL is 5.4. Based on the combined data for all subareas, only 0.68% of children are predicted to have blood-lead levels above 10 µg/dL (Figure 5-11).

FIGURE 5-1

IEUBK Modeling Blood Lead Results - Subarea A

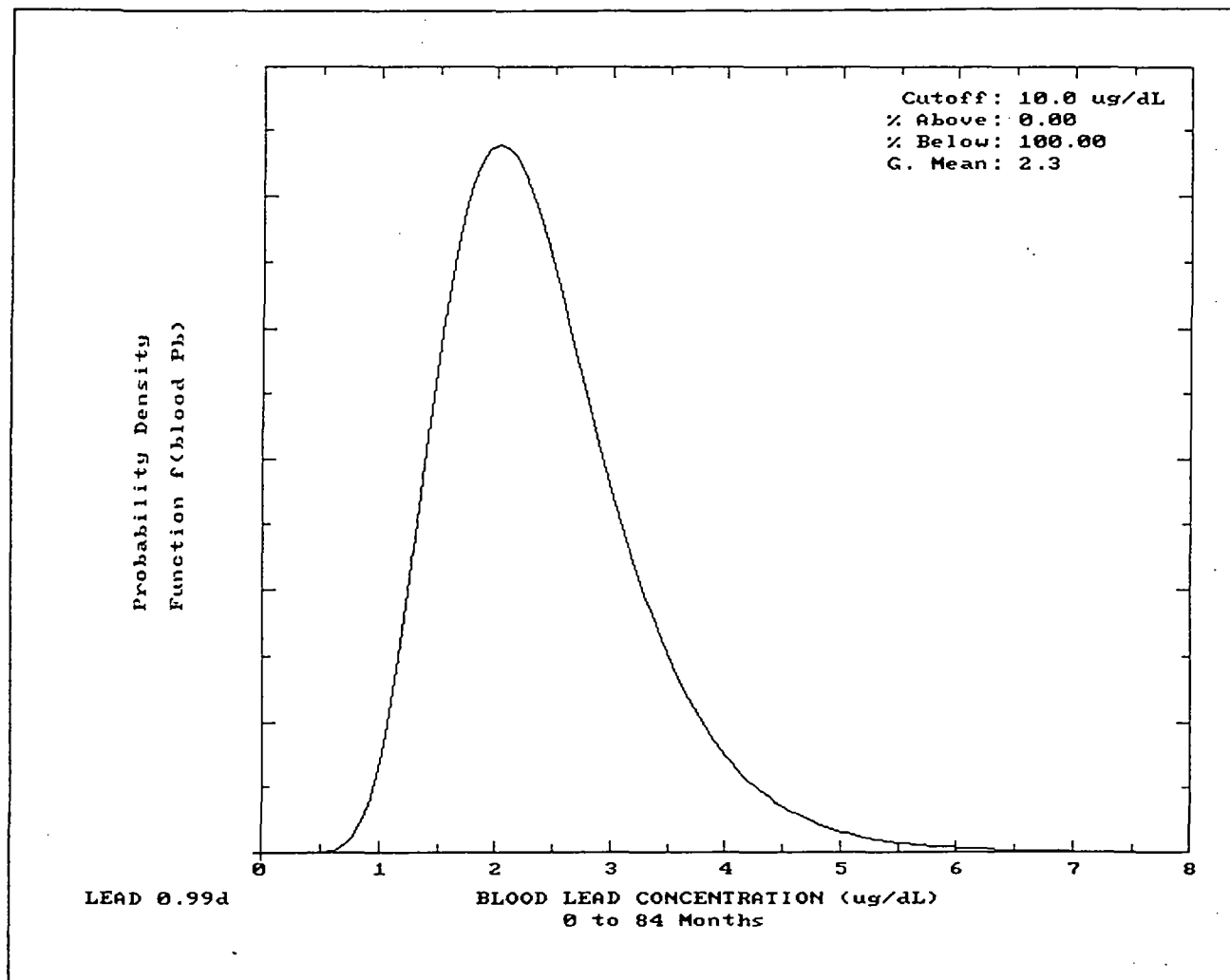


FIGURE 5-2

IEUBK Modeling Blood Lead Results - Subarea B

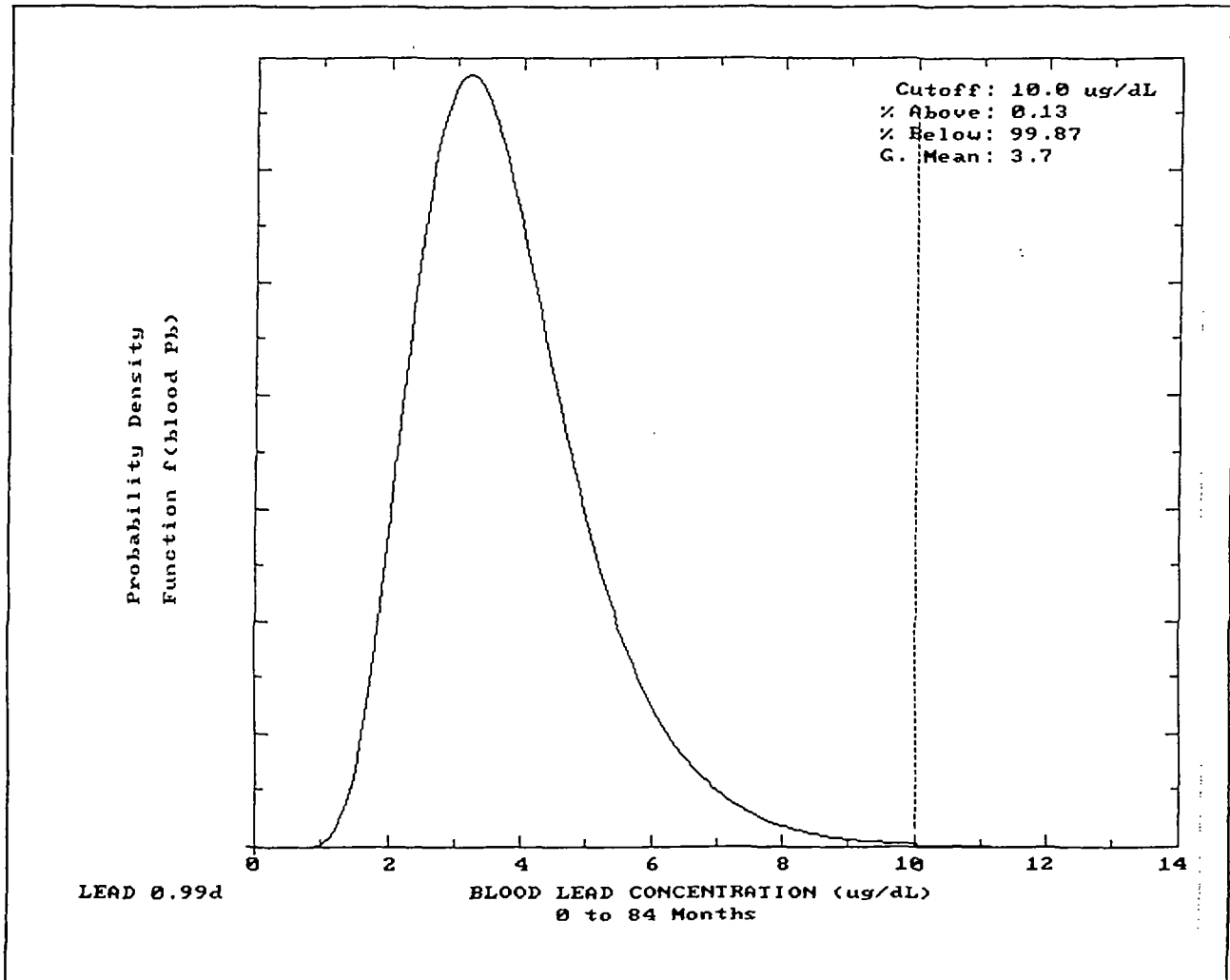


FIGURE 5-3

IEUBK Modeling Blood Lead Results - Subarea C

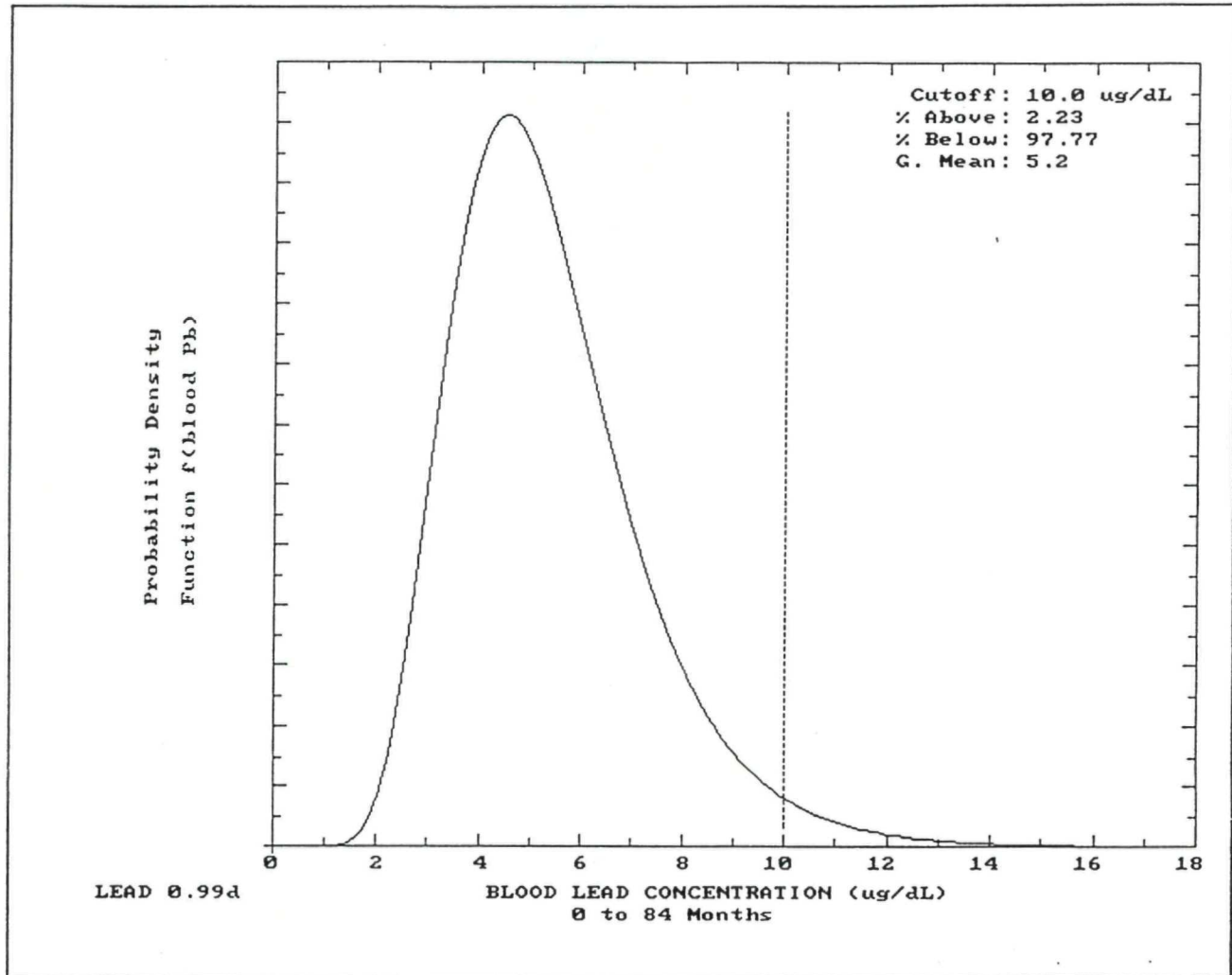


FIGURE 5-4

IEUBK Modeling Blood Lead Results - Subarea D

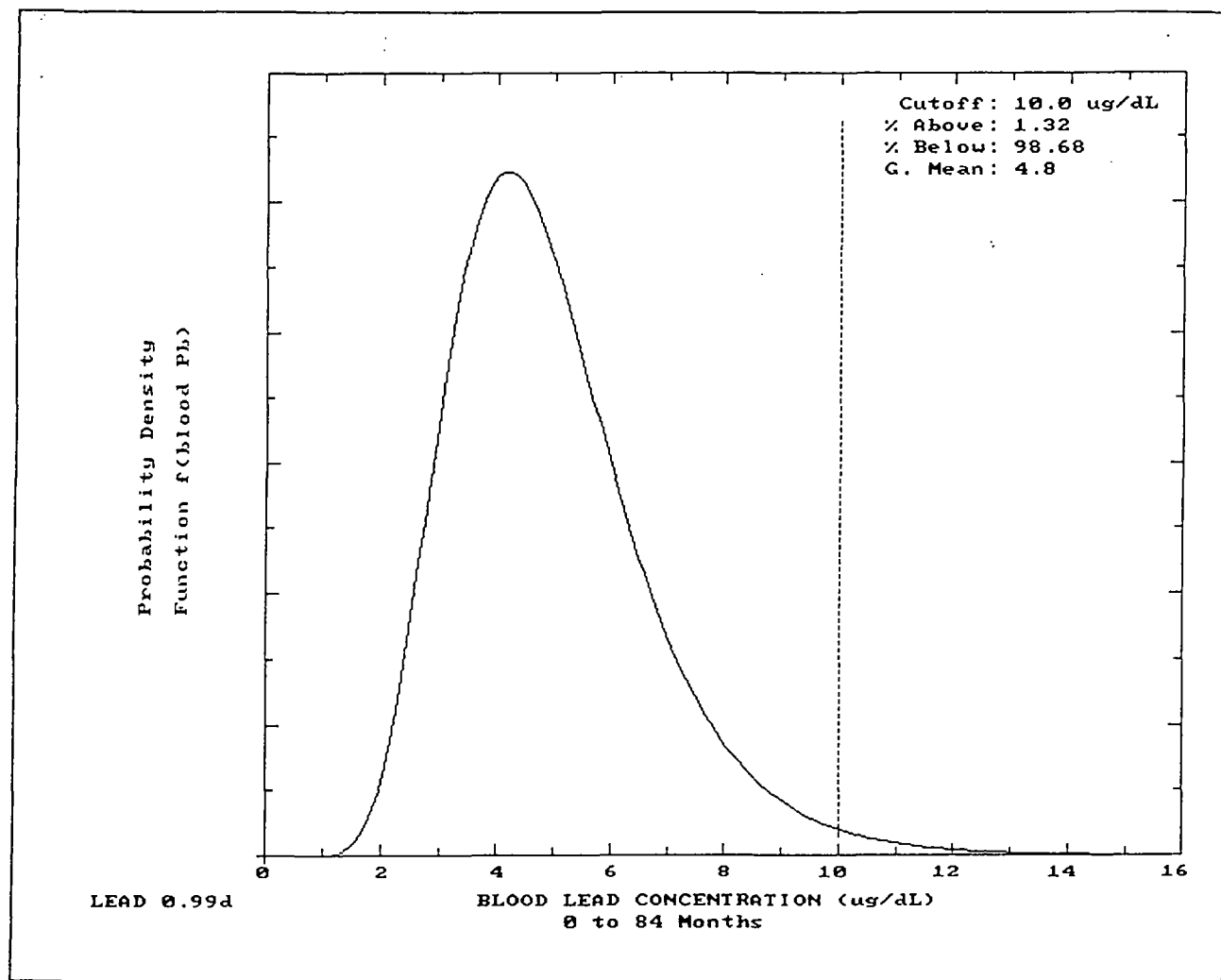


FIGURE 5-5

IEUBK Modeling Blood Lead Results - Subarea E

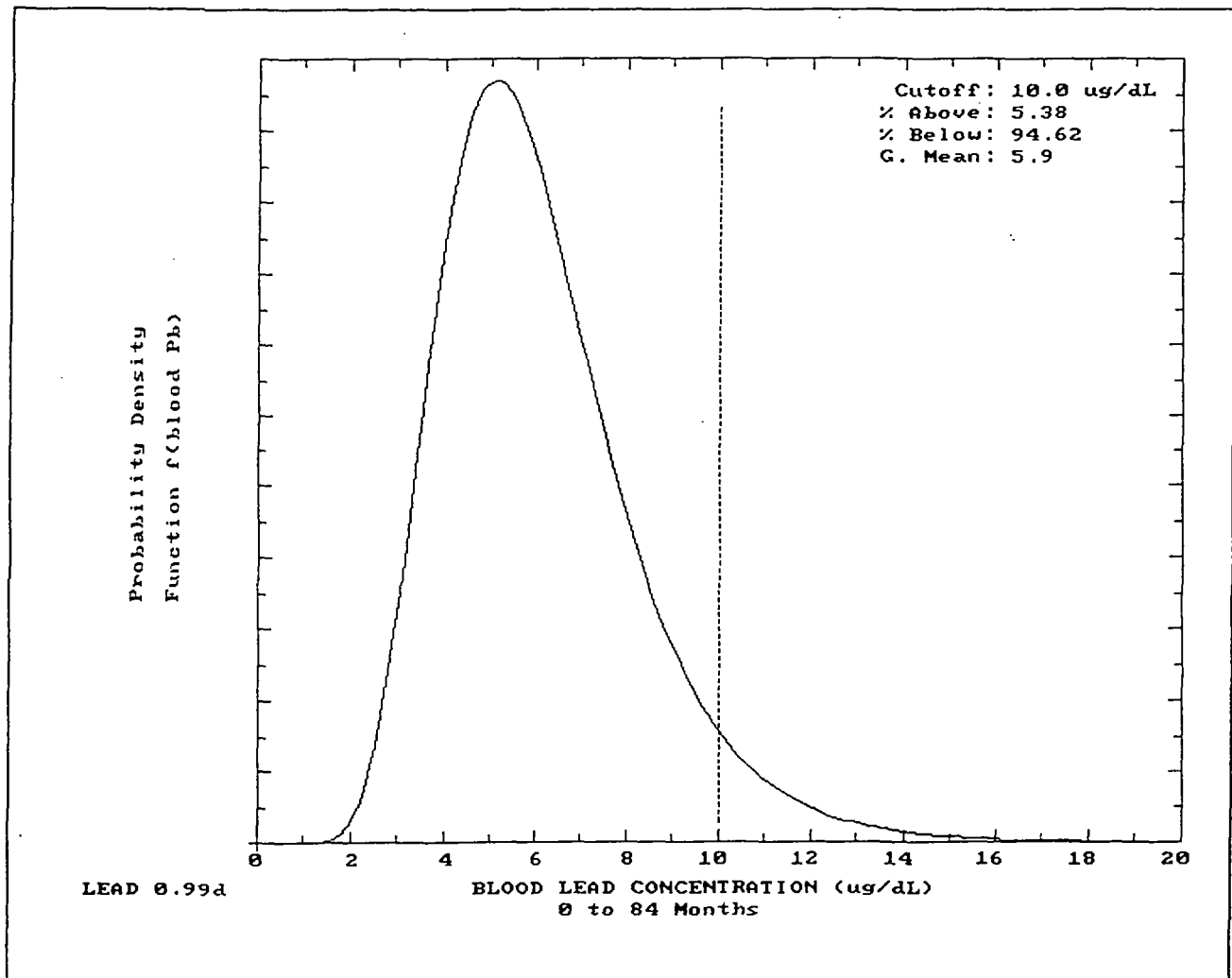


FIGURE 5-6

IEUBK Modeling Blood Lead Results - Subarea F₁

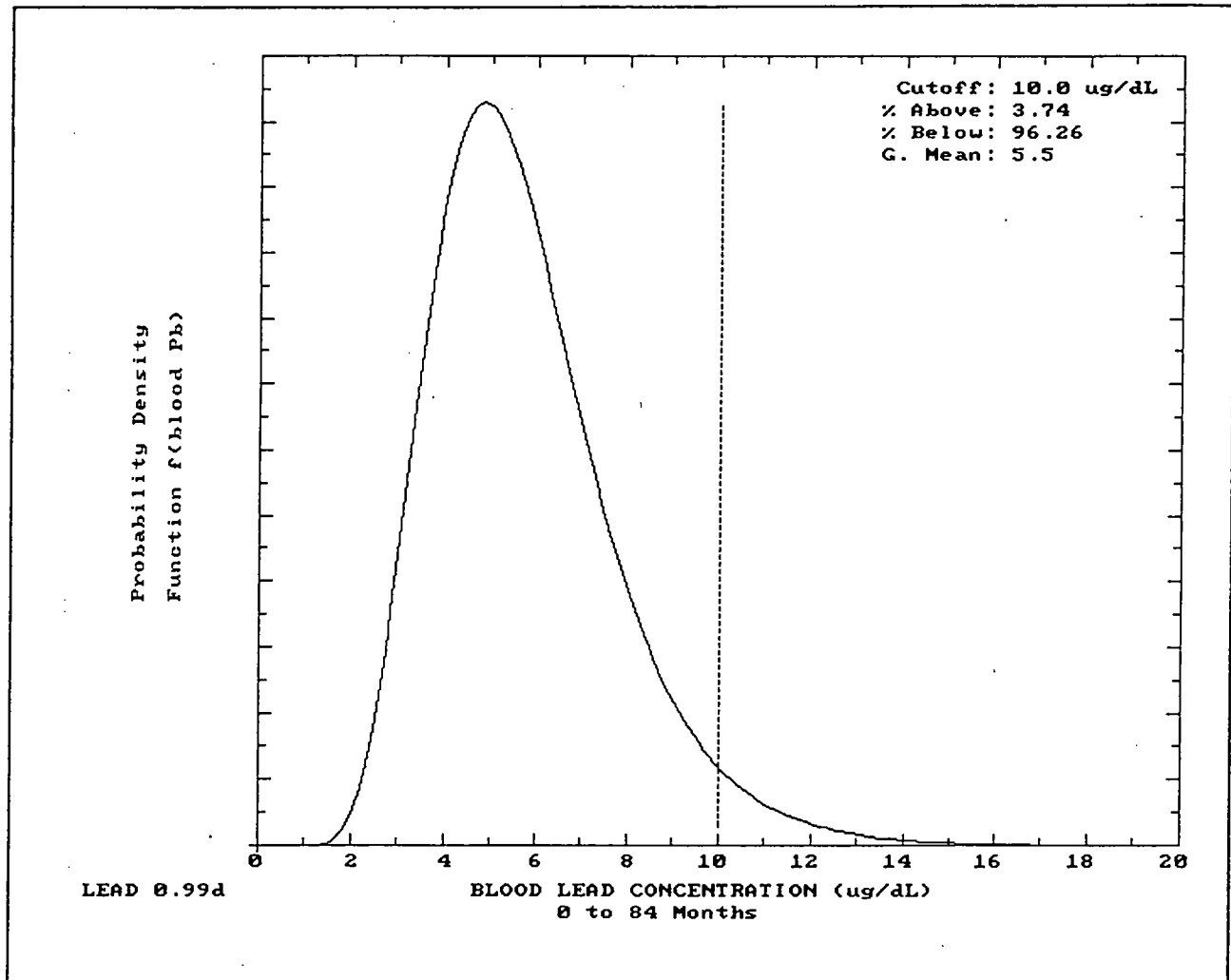


FIGURE 5-7

IEUBK Modeling Blood Lead Results - Subarea F₂

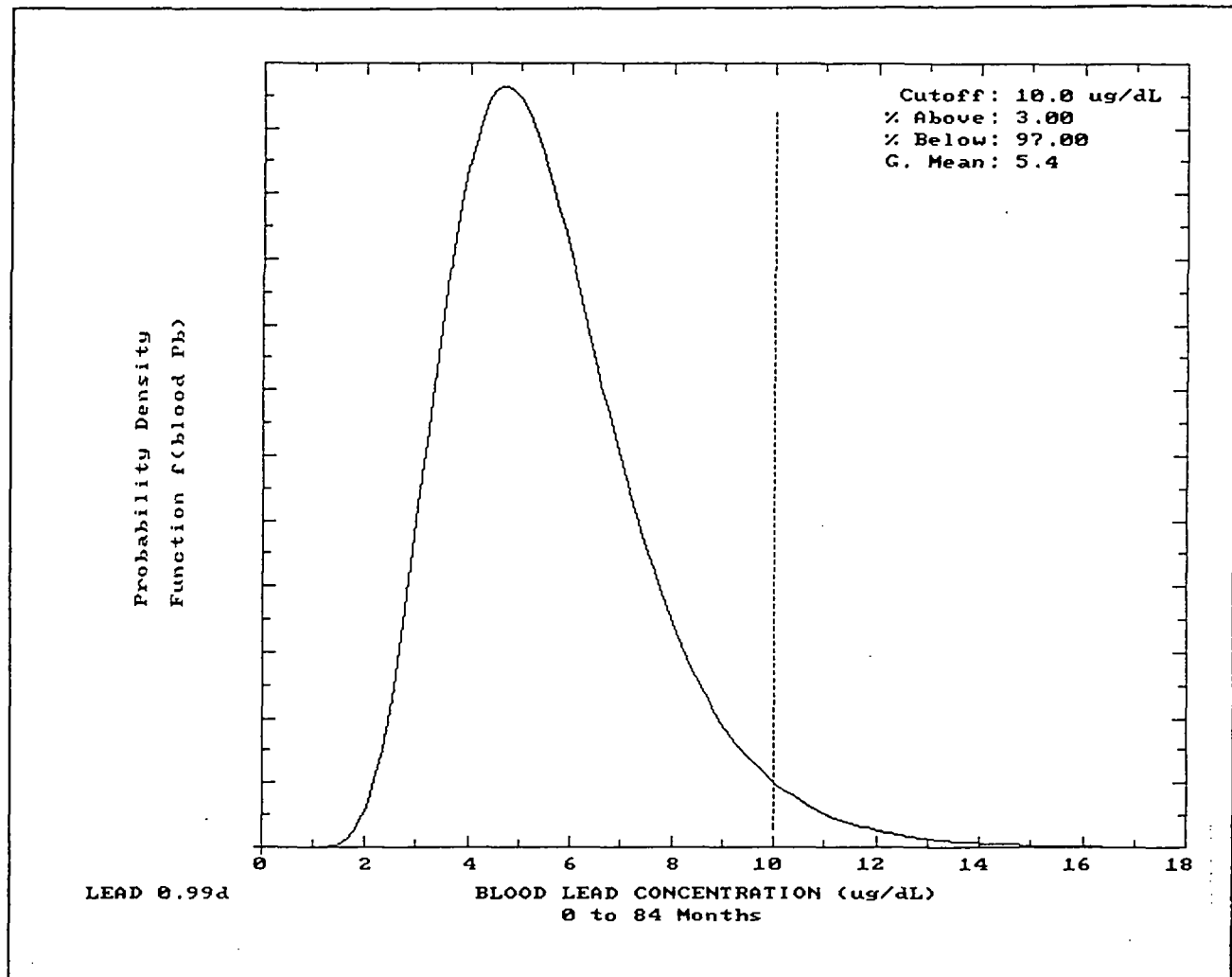


FIGURE 5-8

IEUBK Modeling Blood Lead Results - Subarea I

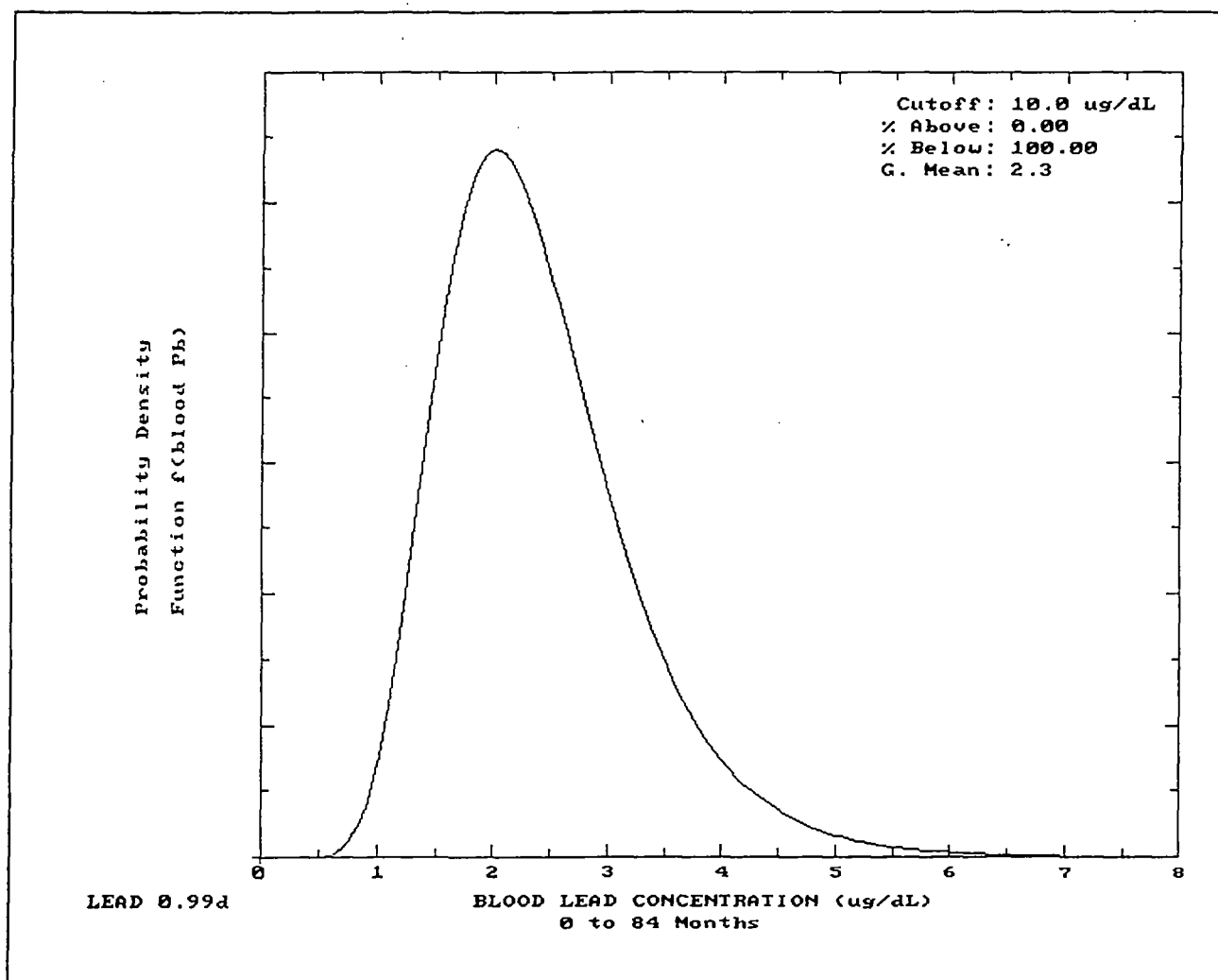


FIGURE 5-9

IEUBK Modeling Blood Lead Results - Subarea J

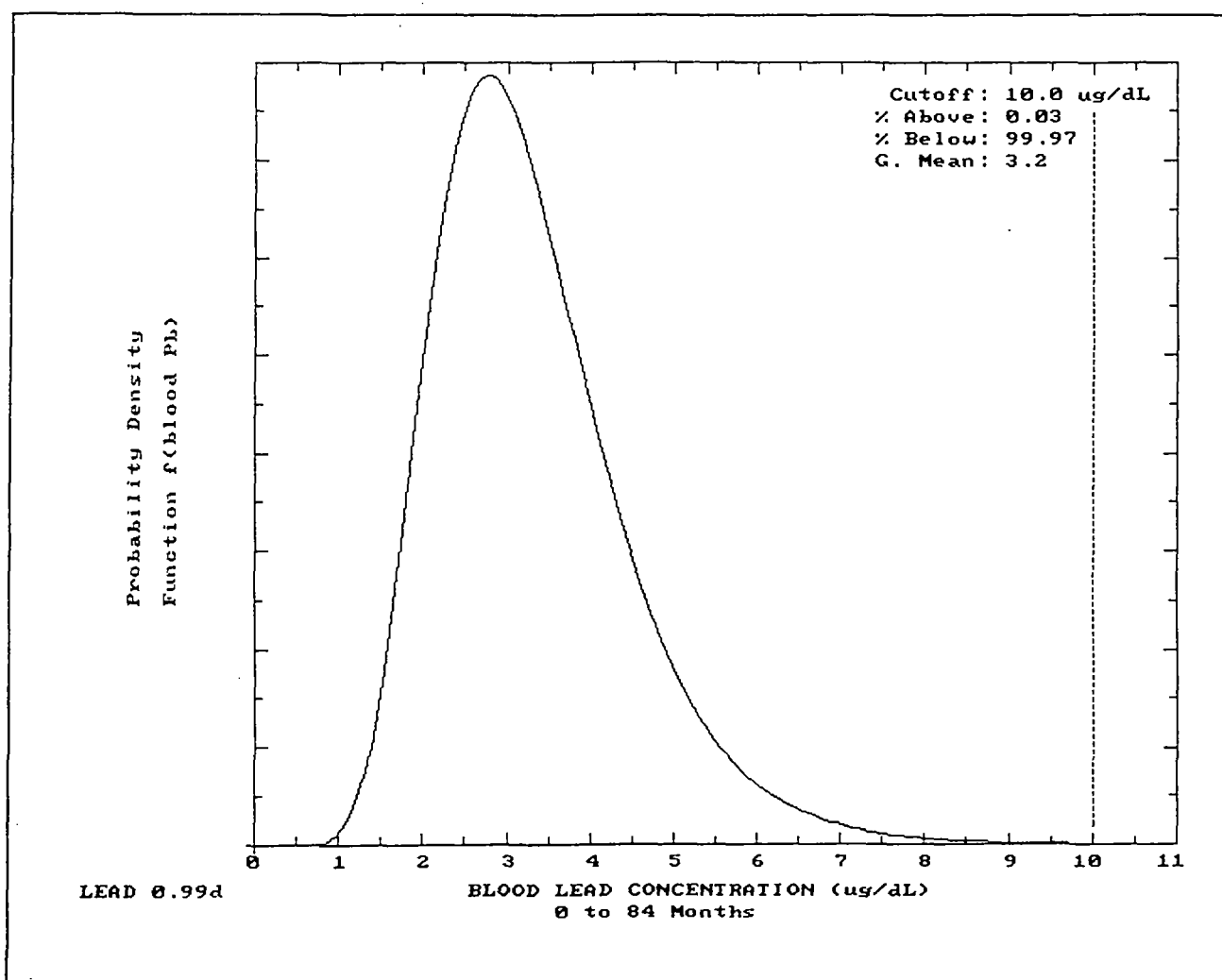


FIGURE 5-10

IEUBK Modeling Blood Lead Results - Subarea G (Opportunity)

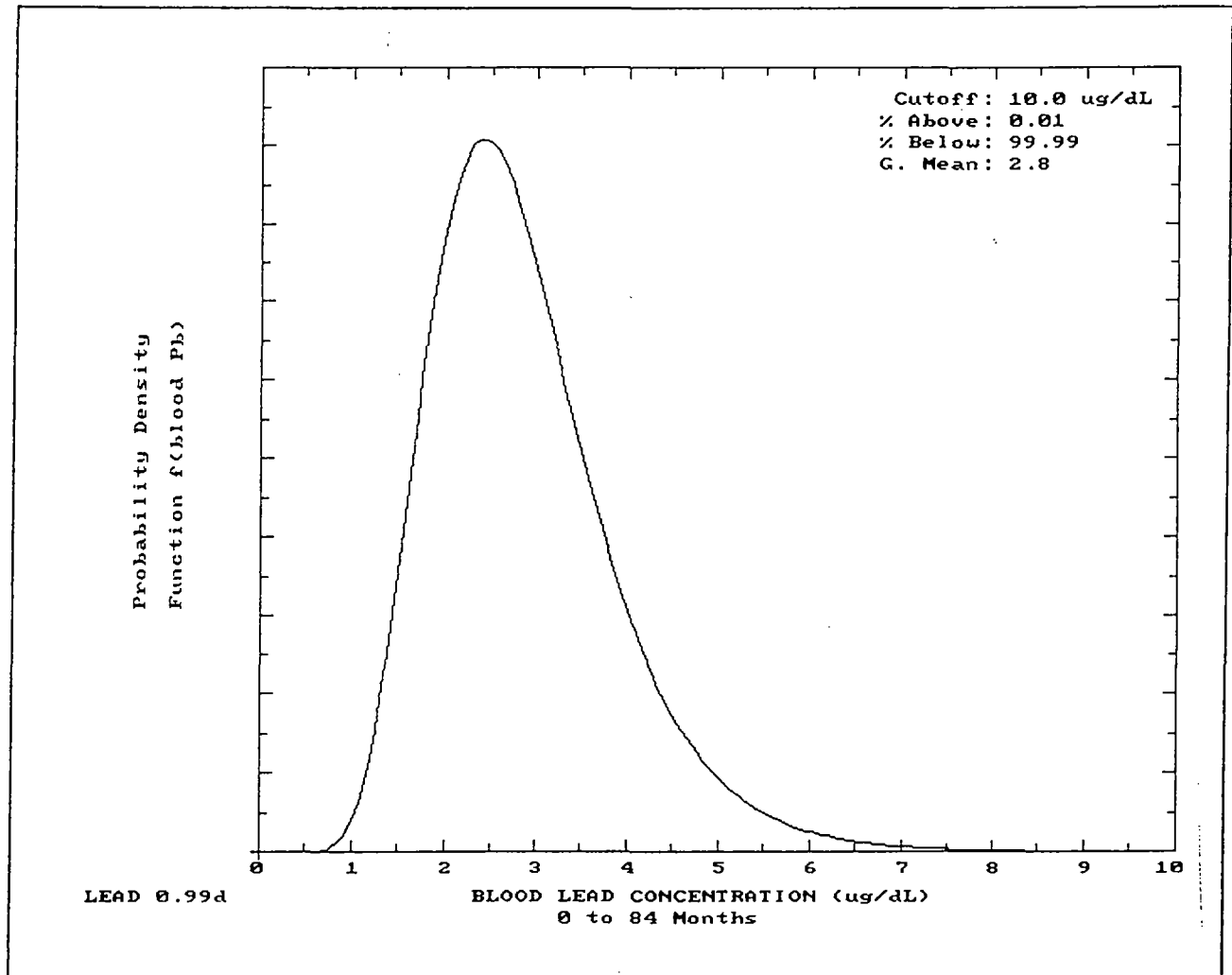


FIGURE 5-11

IEUBK Modeling Blood Lead Results - All Subareas Combined

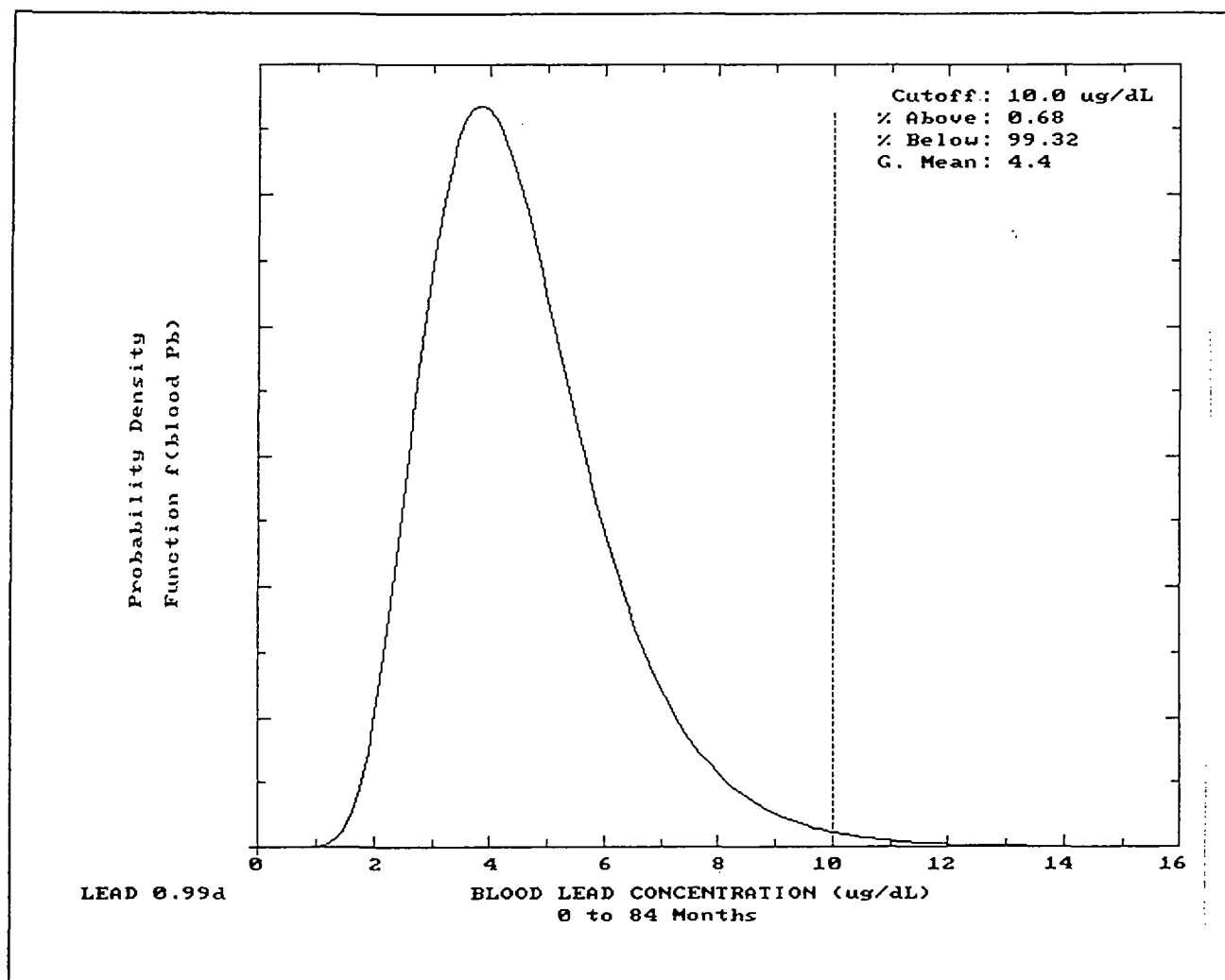


TABLE 5-3

IEUBK Modeling Results Summary

Subarea	Predicted Percentage of Individuals with Blood Lead Levels Above 10 $\mu\text{g/dL}$	Predicted Geometric Mean Blood Lead Level ($\mu\text{g/dL}$)
A	0.00	2.3
B	0.13	3.7
C	2.23	5.2
D	1.32	4.8
E	5.38	5.9
F1	3.74	5.5
F2	3.00	5.4
I	0.00	2.3
J	0.03	3.2
Opportunity	0.01	2.8
All Areas	0.68	4.4

Generally, EPA (1994c) considers risks from exposure to lead unacceptable if more than 5% of children have blood-lead levels in excess of 10 µg/dL. Thus, risk from lead exposure would be considered unacceptable for exposure subarea E.

5.3 ANALYSIS OF UNCERTAINTIES

Quantitative risk estimates are based on site-specific information, national default assumptions, toxicology literature and professional judgement. There are uncertainties associated with all of these sources, and, hence, there is uncertainty in all quantitative estimates of risk. In order to appropriately interpret and use quantitative risk estimates, uncertainties must be recognized and understood. Several sources of uncertainty have been identified in previous sections, including:

- Lack of groundwater data for most of Anaconda
- Limited environmental data for areas outside Anaconda and Opportunity
- Lack of data for lead in interior dust
- Toxicity criteria for arsenic
- Lack of bioavailability data for lead in soils and dust from the study area
- Lack of suitable methodology for evaluating dermal exposure to contaminated soils
- Use of default exposure assumptions and professional judgement in estimating CDI for arsenic and blood-lead levels for lead

In the following discussions, these uncertainties are further evaluated relative to their potential influence on the use of quantitative risk estimates in risk management decisions for the site.

5.3.1 LACK OF DATA FOR CHEMICALS OTHER THAN ARSENIC IN ANACONDA GROUNDWATER

Domestic drinking water samples collected from subarea A in Anaconda were analyzed only for arsenic; therefore, it is possible that other contaminants could be present at significant concentrations. This is not a data gap for Opportunity, since analyses of domestic groundwater in Opportunity did not detect any contaminants other than arsenic at significant concentrations. However, Opportunity is more distant from many waste sources and is characterized by generally lower soil contamination levels than is Anaconda. It may be inappropriate to extrapolate results of groundwater sampling directly from Opportunity to Anaconda.

There are three reasons that suggest that actual effects on the risk assessment are not substantial. First, concentrations of arsenic in subarea A domestic groundwater overlap significantly with background concentrations of arsenic in regional groundwater (Section 2.3.2), indicating that groundwater in this subarea is relatively unimpacted by site contamination.

Second, groundwater downgradient of contamination is not currently used as a source of drinking water within most of the community of Anaconda since municipal water is readily available. Municipal water is supplied by groundwater upgradient of contamination and is unimpacted. Thus, the exposure pathway is incomplete throughout most of the community.

Third, soil levels of cadmium, copper, and zinc were below health-based screening levels, indicating that concentrations in groundwater may be insignificant.

5.3.2 LIMITED ENVIRONMENTAL DATA FOR AREAS OUTSIDE ANACONDA AND OPPORTUNITY

The study area is large, and few data are available for undeveloped or agricultural lands. It is difficult statistically to justify these data as representative, and no quantitative risk estimates are provided outside of Anaconda and Opportunity. Because of the relative sparsity of sampling locations, it is possible that areas with significantly elevated levels of arsenic and lead exist, but have not been recognized. In these areas, risks could be greater than those estimated for Anaconda and Opportunity.

“Hot spot” areas with substantially higher concentrations of contamination are not expected given the primary transport mechanism, air, for contaminants from the smelter and associated wastes. Where data have been collected from more closely spaced locations, results are consistent with a relatively homogeneous distribution of contamination. However, there may be secondary transport mechanisms, such as runoff, which could concentrate contaminants in areas where pooling occurs. Thus, it is theoretically possible that some areas with higher than expected arsenic and/or lead concentrations might exist within the study area. It is not possible to determine the extent of any potential risks that might be associated with such areas. Therefore, screening levels are provided in Section 6.0 for application to areas outside Anaconda and Opportunity. When additional data become available for such areas, screening levels can be used to help assess potential human health risks for current or projected land uses.

5.3.3 LACK OF DATA FOR LEAD IN INTERIOR DUST

No measurements have been made for lead in interior dust for any of the communities within the Anaconda Smelter NPL Site. However, exposures to contaminated dust have been shown to be significant for arsenic in Anaconda and Opportunity, and, in fact, 55% of incidental ingestion of soil-derived contamination is assumed to come via interior dust. In order to

estimate lead concentrations in indoor dust, it was necessary to extrapolate from the available data on arsenic (Bornschein 1992, 1994). This extrapolation required the assumption that arsenic and lead would move in similar fashion from soil to dust, and that there are no significant indoor sources of lead or arsenic that would alter the baseline or "background" concentrations in homes.

The former assumption seems reasonable since one expects that transport from soil to dust will depend mainly on the physical characteristics of soil particles rather than their chemical composition. However, wastes from different sources may have both different particle size distributions and different arsenic and lead concentrations. If such sources contributed differentially to total arsenic and total lead concentrations in the community, there could be differences in transport of arsenic and lead into homes.

The maximum influence different sources might have on dust concentrations would be about a factor of 2 either higher or lower, since concentrations of arsenic in dust are about 43% of those indoors. If lead was transported very efficiently into homes, soil and dust lead concentration might in the worst theoretical case be equal (in several studies there has been no suggestion that soil contaminants might be concentrated in dust). If transport was very inefficient, little or no lead in soil would be transported into homes. Studies at several sites across the country suggest that neither of the above extreme alternatives is likely, implying that any uncertainty in estimates for lead concentrations in dust would be less than a factor of two. This is a relatively small uncertainty and, therefore, lack of interior dust data for lead is not expected to have significant impact on site-related lead exposures².

The baseline or "background" level of lead in indoor dust might be greatly influenced by lead-based paint; a source which would not contribute significantly to interior arsenic contamination. Therefore, the assumption that non-soil sources of arsenic and lead are similar

² This analysis does not consider input from interior lead paint, which can cause indoor dust levels to greatly exceed those in outdoor soils.

is probably incorrect. This assumption, in fact, eliminates consideration of indoor lead-based paint in estimating lead exposures. This may significantly underestimate the potential for exposure in areas where homes have been painted in the past with lead-based paints. Estimates for lead exposure presented in this assessment should not be used to predict potential total lead exposures unless it is known that lead-based paint is not present, or, preferably, measurements for lead in indoor dust have been made.

5.3.4 UNCERTAINTY IN TOXICITY CRITERIA FOR ARSENIC

There continues to be discussion of the oral CSF for arsenic. Recent reviews and letters (Carlson-Lynch, et al. 1994; Beck et al. 1995) present one view of evidence that the oral CSF for arsenic is too high. Several lines of evidence are advanced to support this conclusion, and all are based on criticisms of the studies of the Taiwanese population from which toxicity criteria for ingested arsenic are derived.

First, a recent study (Yost et al. 1994) suggests that estimates of inorganic arsenic in the diet of the Taiwanese population may have been underestimated in the past, resulting in an exaggerated estimate of cancer potency. The study measured inorganic arsenic in rice and sweet potatoes, two staples in the Taiwanese diet, and results were interpreted to indicate that inorganic arsenic in these food stuffs was much greater than previously assumed.

Second, several studies in both humans and laboratory animals were interpreted to indicate that arsenic metabolism is saturable, and that saturation occurs at exposures less than those received by the Taiwanese population. This, in turn, would suggest that the apparent potency of inorganic arsenic as a carcinogen is exaggerated at high doses by reduction in detoxification. At lower doses, efficient metabolism to organic forms would reduce the effectiveness of a given exposure to inorganic arsenic in producing cancer.

Third, inadequate dietary methionine, an essential amino acid, may be present in the Taiwanese diet to support both basic metabolic needs and the metabolic demands caused by the ingestion of large amounts of inorganic arsenic. Methionine is likely to be a methyl donor in the conversion of inorganic arsenic to methylated forms, and lack of sufficient methionine in the diet could limit the capacity for arsenic metabolism in the body. This would result in a higher apparent potency of arsenic, since less metabolic detoxification could take place.

Finally, the presence of humic acids in the water supply for the Taiwanese population is suggested as causative or interactive in the production of human cancer. If humic acids do play such a role, exposure to arsenic in the absence of humic acids may not have the same high potential to cause cancer as that seen in the study population.

Though the above studies seem, on the surface, to make a reasonable case for lowering the arsenic oral CSF, objective examination of all the evidence demonstrates significant flaws in all of the above arguments. An appropriate CSF can only be developed if the limitations of all information is understood and factored into the analysis. On more thorough examination, it does not appear that sufficient information is currently available on which to base a reevaluation of the arsenic CSF.

Data presented by Yost et al. (1994) are dramatically counter to other measurements of inorganic arsenic in rice and potatoes grown in soils treated with inorganic arsenic. This discrepancy is unexplained, but could be due to strong acid treatment used to extract arsenic in the Yost study. This could have resulted in the artifactual production of inorganic arsenic (Mushak and Crocetti 1995). The forms of organic arsenic in plants are poorly known, and it is not clear how easily inorganic arsenic can be produced from these forms, nor how this may vary among different plant species. Until such problems are resolved, it will not be possible to revise the cancer slope factor based on the single least conservative study.

Information available on biotransformation in humans is generally weak and difficult to interpret. Moreover, there are conflicting reports which variously suggest that the saturation point for human methylation of inorganic arsenic falls above or below the exposures received by the Taiwanese population (Mushak and Crocetti 1995). That some reports suggest the former is an important observation. For example, similar percentages for inorganic arsenic, monomethyl arsenic, and dimethyl arsenic were found in urine of subjects in Nevada, exposed on average to levels of arsenic similar to those for the "high dose" group in Taiwan, and in subjects in a control group. The results did not support saturation of metabolism and, in fact, indicated that organic arsenic made up 78 percent of total arsenic in exposed subjects and 86 percent in controls (Warner et al. 1994). Such a small difference is probably not statistically or biologically significant and is not consistent with a low threshold for saturation of arsenic metabolism.

Similarly, in the study by Buchet et al. (1981), which is often cited in support of a relatively low metabolic threshold, data seem to indicate significant metabolic capability at all doses. Individuals receiving 1,000 μg of inorganic arsenic per day, for example, formed nearly the same proportion of total methyl metabolites as did individuals receiving only 125 μg (74 versus 84 percent), respectively. Such differences are small enough to be due to sampling errors and individual variation. On the basis of metabolite formation, it is difficult to conclude that metabolism has reached saturation.

The key to resolving the issue of metabolism in arsenic would seem to be characteristic of mechanisms of methylation and the study of these biochemical pathways in human systems. In addition, empirical studies should focus on the kinetics of the inorganic arsenic rather than on metabolite formation and metabolite ratios. The latter are indirect measures of the amount of the ultimate carcinogen (assumed to be inorganic arsenic) which reaches target tissues. Moreover, metabolite ratios especially are difficult to interpret and have no demonstrated connection with the amounts of inorganic arsenic which reach target tissues.

The nutritional status of the Taiwanese appears to be sufficient for normal metabolic processes (Engel and Receveur 1993). In addition, a simple calculation (Mushak and Crocetti 1995) suggests that the amount of methionine which might be necessary to support metabolism of ingested arsenic is at best a small fraction of total daily intake, on the average of less than 1 percent. It seems likely that the "problem" related to nutritional status is really a "red herring." Until such time as new data become available which challenge the above conclusions, it seems safe to dismiss the argument for nutritional deficits as a factor influencing cancer potency in the Taiwanese populations.

Finally, the presence of humic acids in water consumed by the Taiwanese seems unlikely to be a causative factor in cancer. It appears that arsenic, not humic acids, are the constant in the various stages of both Blackfoot disease and precancerous skin lesions (Mushak and Crocetti 1995). Moreover, both skin cancer and internal cancers are found in patients treated with Fowler's solution where humic acids were not a factor (EPA 1986). Thus, it has been reasonably concluded that humic acids are not necessary for the carcinogenic activity of arsenic. It is possible that humic acids could alter the carcinogenic response in humans through some as yet unknown mechanism. Available data are, however, apparently not sufficient to establish this as a possibility, much less quantify such an effect. Until substantial additional data are available, it will not be possible to assess the contribution, if any, of humic acids to carcinogenesis in the Taiwanese population.

Currently, regional guidance recommends recognizing uncertainties in the arsenic oral CSF, but making no changes in the CSF for the purposes of quantitative risk assessment, and this approach is taken in this assessment. It is, thus, assumed that uncertainties in the arsenic oral CSF are best taken into account in the risk management process.

5.3.5 LACK OF DATA ON BIOAVAILABILITY OF LEAD IN SOILS AND INTERIOR DUST

Bioavailability of lead in wastes from mining and smelting activities is recognized as an important factor in human exposure. In some cases, such as Butte, Montana, bioavailability of lead has been shown to be much reduced from the default value of 30 percent in the IEUBK model. These findings in animal studies are consistent with a large exposure study of young children living in Butte (Bornschein 1991).

Some wastes in Anaconda, such as the mill tailings in the Opportunity Ponds, may be similar to wastes found in Butte. These wastes were both derived from the same area and from similar processes. However, other wastes in Butte (e.g., waste rock) are not found in Anaconda. Further, large quantities of lead may have been released from the smelter in Anaconda, whereas only limited smelting occurred in Butte. Due to these differences, it is not possible to extrapolate bioavailability estimates from Butte to Anaconda.

Correlations with other studies can often be drawn based on geochemistry in soil. However, no speciation data are available to determine the dominant forms of lead in soils at the Anaconda site. Without such data, one must consider measurements of high bioavailability of lead in wastes at the Smuggler Mine NPL site in Colorado, and the Jasper County, Missouri NPL site (Griffin 1995) where considerable lead smelting occurred, and the blood lead studies in East Helena, Montana (EPA 1986). Such studies support bioavailability estimates at or above the model default of 30 percent (unpublished results from swine bioavailability studies show absolute bioavailabilities in excess of 40% for the Smuggler Mine and Jasper County sites).

It is conceivable that bioavailability of lead in soils at Anaconda is less than the IEUBK model default. However, site-specific data on which to base a quantitative estimate of bioavailability are lacking. Thus, it is reasonable to assume that the default assumption in the

IEUBK model is protective. It is not possible to determine at this time, however, if the default is overly conservative.

5.3.6 LACK OF METHODOLOGY FOR EVALUATING DERMAL EXPOSURE TO METALS IN SOIL

Little information is available on which to base estimates of dermal absorption of arsenic or metals in soil. However, a few reports suggest that absorption will be very inefficient, even for soluble arsenic or metal forms (ATSDR 1991a). This information is difficult to extrapolate directly to human exposure, but it does suggest that dermal exposure should be a minor pathway. It is not likely that dermal absorption of arsenic or metals from soil contributes significantly to overall exposures within the study area.

5.3.7 USE OF DEFAULT EXPOSURE ASSUMPTION AND PROFESSIONAL JUDGEMENT

Default exposure assumptions and professional judgement are used throughout the exposure assessment to estimate potential chronic daily intakes. Data are not available to determine quantitatively how each of these assumptions and judgements might influence CDI calculations. However, as discussed in Section 3.4, urinary arsenic concentrations predicted using the basic assumptions also used in the exposure assessment are in good agreement with those actually measured in the community of Anaconda. This suggests that assumptions and judgements made are reasonable and uncertainty in the results of the exposure assessment is relatively small, at least for young children.

It should be also be noted that uncertainties in exposure assumptions not directly assessed by the comparison of observed and predicted urinary arsenic in children are not expected to greatly influence exposure estimates. As discussed in section 3.5, factors such as soil/dust ingestion rates for adults, and exposure frequency and duration, are at least conservative (i.e.,

are unlikely to underestimate possible exposures) and probably do not result in substantial overestimation.

It is reasonable to conclude that exposures calculated in this assessment are acceptable for both CTE and RME estimates.

5.4 SUMMARY

Section 5.1 presents cancer and noncancer risks associated with exposure to arsenic in groundwater, soil, and dust. Cancer risks based on RME for soil/dust for all subareas of the site evaluated fall into a narrow range of about 1×10^{-5} to 3×10^{-5} . This narrow range reflects the relatively even distribution of arsenic within Anaconda and Opportunity. A similar narrow range of hazard quotients (0.1 to 0.3) is estimated for the same exposures. Cancer risk estimates all fall within the EPA risk range, and all hazard quotients fall below the target level of one.

In subarea A and in Opportunity, cancer risks based on RME for groundwater are in the same range as those for exposure to soil/dust; this is also true for hazard quotients. Again, all estimates are within acceptable ranges, or below risk targets.

Combined risks and hazard quotients from RME to both groundwater and soil/dust (subarea A and Opportunity only) remain within the ranges of risks and HQs established for RME for soil/dust ingestion. This suggests that even where near maximum exposures to both groundwater and soil/dust occur simultaneously, exposures will remain within acceptable levels.

EPA (1994c) generally considers risks from lead exposure unacceptable if more than 5% of children have blood-lead levels exceeding $10 \mu\text{g/dL}$. Five percent of the children in subarea E are estimated to have blood-lead levels exceeding $10 \mu\text{g/dL}$.

Analysis of uncertainties in the above risk estimates suggests that it is unlikely that risks have been significantly underestimated, especially for the well characterized communities of Anaconda and Opportunity. The sparsity of data in other areas, however, leaves open the question of whether significant "hot spots" might exist within the study area. In the absence of data, screening levels are provided in Section 6.0 to assist in assessing specific locations when additional data become available.

6.0 RISK-BASED SCREENING LEVELS

This section of the HHRA develops risk-based screening levels for the Anaconda Smelter NPL Site. Screening levels are developed for arsenic in soil and surface water, and are based on exposure assumptions for residential, agricultural, occupational, and recreational exposure scenarios. Exposure assumptions for agricultural, occupational, and recreational scenarios are taken from other risk assessments prepared for the Anaconda Smelter NPL Site or adjacent areas. Screening levels are intended to be used together with site maps that illustrate chemical concentration boundaries (generated using the kriging technique) to identify potential areas of risk. Also, screening levels can be used to evaluate any additional data collected from specific locations. Finally, comparison of screening levels for scenarios not evaluated quantitatively in this HHRA provides further evidence regarding the potential significance of exposures associated with non-residential land use. Such comparisons may provide useful information pertinent to evaluation of remedial options for different areas of the site.

Screening levels are developed for the following scenarios:

- Resident
- Agricultural worker (exposure during plowing/tilling)
- Commercial worker
- Recreational visitor (dirt-bike rider)
- Recreational visitor (adolescent playing in pooled water)

Screening levels for soil are developed based on the following exposure pathways:

Residents (Adults and Children Ages 0 - 6):

- Ingestion of surface soils
- Ingestion of interior dust

Agricultural Workers (Adults):

- Ingestion of surface soils
- Inhalation of dust

Recreational Users (Dirt Bike Riders):

- Ingestion of surface soils
- Inhalation of dust

Recreational Visitors (Swimmers/Waders):

- Ingestion of surface water
- Dermal exposure to surface water

Commercial Workers (Adults):

- Ingestion of surface soils
- Ingestion of interior dust

Screening levels are developed based on the carcinogenic and noncarcinogenic effects of arsenic. Screening levels are developed for a carcinogenic risk range of 10^{-3} to 10^{-7} , and a noncarcinogenic HI of 1. Screening levels are developed for both RME and CTE exposure scenarios.

6.1 EXPOSURE ASSUMPTIONS

6.1.1 RESIDENTIAL EXPOSURE SCENARIO

Screening levels for the residential exposure scenario are calculated using the exposure assumptions used in this HHRA to evaluate residential exposures at the Anaconda Smelter NPL Site. Screening levels for this exposure scenario are based on ingestion of surface soil/interior dust. Exposure assumptions for the residential scenario are presented in Table 6-1 and are discussed below. Screening levels for residential exposures are calculated according to the following formulas:

TABLE 6-1

Exposure Parameters for the Residential Scenario

Symbol	Units	Definition	Value	Source
SL	(mg arsenic/kg soil)	risk-based screening level	Section 6-2	-
TR	(unitless)	target risk	Section 6-2	-
AT	(days)	averaging time	Carcinogens = 25,550 Noncarcinogens RME = 10,950 CTE = 3,285	EPA 1989a
CF	(kg/mg)	conversion factor	.000001	EPA 1989a
EF	(days/year)	exposure frequency	350	EPA 1989a
SF _a	(mg/kg-day) ⁻¹	oral slope factor for arsenic	1.5	EPA 1995b
IR _{child}	(mg/day)	soil ingestion rate for children	RME=200 CTE=100	EPA 1993a EPA 1993a
ED _{child}	(years)	exposure duration for children	RME=6 CTE=2	EPA 1993a EPA 1993a
BW _{child}	(kg)	average body weight for children	15	EPA 1989a
IR _{adult}	(mg/day)	soil ingestion rate for adults	RME=100 CTE=50	EPA 1993a EPA 1993a
ED _{adult}	(years)	exposure duration for adults	RME=24 CTE=7	EPA 1993a EPA 1993a
BW _{adult}	(kg)	average body weight for adults	70	EPA 1989a
FS	(unitless)	fraction of soil ingested	0.45	Professional Judgement
BAF _s	(unitless)	bioavailability of soil	0.183	EPA 1995a
C	(unitless)	Contribution of soil arsenic to arsenic in dust	0.43	Calculated, see text.
FD	(unitless)	fraction of dust ingested	0.55	Professional Judgement
BAF _D	(unitless)	bioavailability of interior dust	0.258	EPA 1995a

For carcinogenic exposures:

$$SL = \frac{(TR \times AT)}{(CF \times EF \times SF_o) \left(\frac{IR_{child} \times ED_{child}}{BW_{child}} + \frac{IR_{adult} \times ED_{adult}}{BW_{adult}} \right) \times [(FS \times BAF_s) + (C \times FD \times BAF_D)]}$$

For noncarcinogenic exposures:

$$SL = \frac{(TR \times AT \times RfD)}{(CF \times EF) \times \left(\frac{IR_{child} \times ED_{child}}{BW_{child}} + \frac{IR_{adult} \times ED_{adult}}{BW_{adult}} \right) \times [(FS \times BAF_s) + (C \times FD \times BAF_D)]}$$

Where:	SL	=	Screening Level for Soil (mg/kg)
	TR	=	Target Risk (unitless)
	AT	=	Averaging Time (days)
	RfD	=	Reference Dose for Arsenic (mg/kg-day)
	CF	=	Conversion Factor for Soil (kg/mg)
	EF	=	Exposure Frequency (days/year)
	SF _o	=	Oral Slope Factor for Arsenic (mg/kg-day ⁻¹)
	IR	=	Soil Ingestion Rate (mg/kg)
	ED	=	Exposure Duration (years)
	BW	=	Body Weight (kg)
	FS	=	Fraction of Soil Ingested (unitless)
	BAF _s	=	Bioavailability Factor for Soil (unitless)
	C	=	Contribution of Soil Arsenic to Arsenic in Dust (unitless)
	FD	=	Fraction of Dust Ingested (unitless)
	BAF _D	=	Bioavailability Factor for Dust (unitless)

Averaging Time

Carcinogenic exposures are averaged over 70 years or 25,550 days (EPA 1989a). AT for noncarcinogenic exposures is equal to ED x 365 days/year. AT for RME noncarcinogens is, therefore, 10,950 days and AT for CTE is 3,285 days.

Exposure Frequency

For residents, EF is assumed to be 350 days/year (EPA 1989a). This value assumes that residents will take a single two-week vacation per year. An EF of 350 days/year is used to evaluate soil and dust ingestion by residents. This value is conservative, since soil in

Montana is likely to be frozen for several months out of the year, and ingestion of soil during this time is unlikely.

Ingestion Rate

Soil/dust IRs for young children (0 to 6 years old) and adult residents are 200 and 100 mg/day for RME, and 100 and 50 mg/day for CTE (EPA 1993a). Time-weighted CTE and RME soil/dust IR are calculated assuming 6 years of exposure for children and 24 years for adults.

Exposure Duration

The EPA (1993a) recommended ED for RME is 30 years for residents, spanning a time period from birth into adulthood. Exposures are time-averaged over 30 years, assuming 6 years of exposures for children and 24 years for adults. For evaluation of CTE, EPA (1993a) recommends using EDs of two years for children and 7 years for adults for a total ED of nine years.

Body Weight

The default BW parameters for young children and adults are 15 and 70 kg, respectively (EPA 1989a). These values are used to calculate screening levels based on residential exposures.

Fraction of Soil and Dust Ingested

Of the total amount of soil/dust ingested (see Ingestion Rate above), 45% is assumed to come from soil and 55% from interior dust.

Bioavailability of Soil and Dust

The values selected for soil and dust bioavailability (BAF_s and BAF_D) were derived by EPA (1994a, 1995a) from data presented in "Determination of Bioavailability of Soluble Arsenic and Arsenic in Soil and Dust Impacted by Smelter Activities Following Oral Administration

in Cynomolgus Monkeys" (Battelle 1994). The selected values (18.3% for soil and 25.8% for dust) are the mean absolute bioavailabilities identified in this study.

Contribution of Soil Arsenic to Arsenic in Dust

The contribution of soil arsenic to arsenic in dust was derived from multiple regression analyses performed by Bornschein (1994) on data collected during the Anaconda arsenic exposure study. This is described in detail in Section 3.3.1.2.

6.1.2 AGRICULTURAL WORKER SCENARIO

Screening levels for soil are developed for the agricultural worker scenario for the exposure pathways of soil ingestion and inhalation of windblown dust. Agricultural workers at the site could come into contact with contaminants in soils during plowing, planting, field maintenance, and harvesting, and incidental ingestion of soil and inhalation of dust may result in significant exposure. Incidental ingestion of soil may occur when contaminated soil adheres to hands and is subsequently ingested via hand-to-mouth behavior (e.g., when eating lunch), and dust may be inhaled when it is resuspended during field work.

Exposure assumptions used for the agricultural worker scenario are derived from the Draft Baseline Risk Assessment for the SST OU, Silver Bow Creek NPL Site (CDM Inc. 1994) and from relevant EPA guidance documents. Exposure assumptions are summarized in Table 6-2 and are discussed below. Screening levels for soil based on the agricultural worker scenario are calculated using the following formula based on carcinogenic exposures:

$$SL = (TR \times AT \times BW) / ((EF \times IR_s \times ED \times CF_s \times SF_o \times BAF_s) + (EF \times ED \times IR \times SF_i \times DL \times ET))$$

Where:

SFi	=	Inhalation Slope Factor for Arsenic (mg/kg-day)
DL	=	Dust Loading Factor (kg/m ³)
ET	=	Exposure Time (hours/day)

All other parameters are previously defined.

TABLE 6-2

Exposure Parameters for the Agricultural Worker Scenario

Symbol	Units	Definition	Value	Source
SL	(mg arsenic/kg soil)	risk-based screening level	Section 6-2	-
TR	(unitless)	target risk	Section 6-2	-
AT	(days)	averaging time	25550	EPA 1989a
BW	(kg)	body weight	70	EPA 1989a
EF	(days/year)	exposure frequency	RME = 140 CTE = 84	Site-specific Site-specific
ED	(year)	exposure duration	RME = 30 CTE = 9	EPA 1989a EPA 1989a
IRs	(mg/day)	soil ingestion rate	RME = 480 mg/day for 14 days, 100 mg/day for 126 days CTE = 100 mg/day for 14 days, 50 mg/day for 70 days	EPA 1993a Professional Judgement
CFs	(kg/mg)	conversion factor for soil	0.000001	EPA 1989a
SFo	(mg/kg-day) ⁻¹	oral slope factor for arsenic	1.5	EPA 1995b
BAF _s	(unitless)	bioavailability of soil	0.183	EPA 1995a
IR	(m ³ /hour)	inhalation rate	2.5	EPA 1989b
SFi	(mg/kg-day) ⁻¹	slope factor for inhalation	15	EPA 1995b
DL	(kg/m ³)	dust loading factor	RME = 1.5×10^{-7} kg/m ³ for 14 days, 2.2×10^{-10} kg/m ³ for 126 days CTE = 1.5×10^{-7} kg/m ³ for 14 days, 2.2×10^{-10} kg/m ³ for 70 days	Professional Judgement
ET	(hours/day)	exposure time	8	Site-specific

Screening levels based on noncarcinogenic exposure cannot be calculated for the agricultural worker scenario. The exposure pathways evaluated for this scenario are ingestion of soil and inhalation of dust. However, a reference concentration that can be used to evaluate noncarcinogenic inhalation exposure is not available for arsenic. Screening levels based on noncarcinogenic exposure are, therefore, not calculated for this scenario.

Averaging Time

Carcinogenic exposures are averaged over 70 years or 25,550 days (EPA 1989a). This value is used to calculate screening levels based on exposure by agricultural workers.

Body Weight

The default BW parameters for adults is 70 kg, respectively (EPA 1989a). This value is used to calculate screening levels based on the agricultural worker scenario.

Exposure Frequency

To calculate screening levels for the agricultural worker scenario, CTE and RME EFs of three and five days per week, respectively are assumed. It is also assumed that ingestion of soil is not possible at times when the soil is frozen (approximately five months each year).

Exposures are therefore only evaluated for seven months out of the year, and EFs of 140 and 84 days per year are assumed to calculate screening levels based on RME and CTE exposure, respectively. These EFs may be high, as agricultural workers may not be engaged in activities during which they could contact soil each working day.

Exposure Duration

EPA (1991c) recommends using an ED of 25 years to evaluate worker exposures. However, most agricultural workers near the Anaconda Smelter are expected to also be residents of the area. Residential EDs are, therefore, considered more appropriate for evaluation of these workers. For development of screening levels based on RME, an ED of 30 years is used, which is an upper range (90th percentile) estimate for residency at one address (EPA 1989a).

Screening levels for CTE are based on an ED of nine years, the average estimate for residency at one address (EPA 1989a).

Soil Ingestion Rate

EPA (1993a) recommends a soil IR of 480 mg/day to evaluate RME from soil ingestion for professions during which intensive contact with soil may occur. For agricultural worker exposures at the Anaconda Smelter NPL Site, intensive contact with soil is only expected during plowing. Based on the types of crops grown at the site, plowing is assumed to be limited to a maximum of 14 days per year. During non-contact intensive activities (e.g., watering, planting, and harvesting), lower soil IR are considered more appropriate. For such activities the EPA (1993a) default soil IR of 100 mg/day for workers is used for the RME. The EF for RME for agricultural workers is 140 days/year. To calculate screening levels based on RME, it is assumed that farmers ingest 480 mg of soil for 14 days per year and 100 mg/day for 126 days per year.

Current data are insufficient to estimate a CTE soil ingestion rate for workers who may have intensive contact with soil (i.e., during plowing) (EPA 1993a). Based on professional judgement, an average soil IR of 100 mg/kg is considered reasonable for such exposures. This value is used for CTE soil ingestion during plowing. The default soil IR for activities associated with non-intensive contact with soil (50 mg/day) (EPA 1991c) is used for CTE during agricultural activities other than plowing. The EF for CTE worker exposures is 84 days/year. Screening levels based on CTE are calculated assuming a soil IR of 100 mg/day for 14 days per year and 50 mg/day for 70 days per year.

Bioavailability of Soil

The BAF_s of 18.3% was derived by EPA (1994a, 1995a) from data presented in "Determination of Bioavailability of Soluble Arsenic and Arsenic in Soil and Dust Impacted by Smelter Activities Following Oral Administration in Cynomolgus Monkeys" (Battelle 1994), and presents the mean absolute bioavailability identified in this study.

Inhalation Rate

Agricultural workers are assumed to be moderately active when working in the fields. EPA (1989b) presents an inhalation rate of 2.5 m³/hour for adults at moderate activity levels. This inhalation rate is used here.

Dust Loading Factor

As for soil ingestion (see above), quantities of dust inhaled are assumed to be highest during plowing and relatively lower during other activities generally not associated with intensive contact with soil. Two dust loading factors are, therefore, estimated, one for plowing and one for other agricultural activities. A particulate emission factor that estimates the concentration of respirable particles that may be present in the air, due to wind erosion, was derived by Cowherd, et al. (1985) and is presented by EPA (1991a). The estimated value is based on the assumptions that the surface material has unlimited erosion potential, and would erode at low wind speed. These assumptions result in a conservative estimate for particulate intake. Cowherd, et al. (1985) used the following formula to derive the particulate emission factor:

$$PEF (m^3/kg) = \frac{LS \times V \times DH \times 3600 \text{ s/hr}}{A} \times \frac{1000 \text{ g/kg}}{0.036 \times (1-G) \times (U_m/U_t)^3 \times F(x)}$$

Where:

PEF	=	Particulate emission factor (m ³ /kg) (default = 4.63 x 10 ⁹ m ³ /kg)
LS	=	Width of contaminated area (m) (default = 45 m)
V	=	Wind speed in mixing zone (m/s) (default = 2.25 m/s)
DH	=	Diffusion height (m) (default = 2 m)
A	=	Area of contamination (m ²) (default = 2025 m ²)
0.036	=	Respirable fraction (g/m ³ -hr) (default 0.36 g/m ³ -hr)
G	=	Fraction of vegetative cover (unitless) (default = 0)
U _m	=	Mean annual wind speed (m/s) (default = 4.5 m/s)
U _t	=	Equivalent threshold value of wind speed at 10 m (m/s) (default = 12.8 m/s)
F(x)	=	Function dependent on U _m /U _t (unitless) (default = 0.0497 [determined by Cowherd, et al. 1985])

The resulting value, $4.63 \times 10^9 \text{ m}^3/\text{kg}$ or $2.2 \times 10^{-10} \text{ kg/m}^3$ is the dust loading factor that is used to evaluate exposure associated with agricultural activities such as watering and planting. This value is used to evaluate exposure for 126 days and 70 days for RME and CTE exposure, respectively.

Dust loading while plowing has not been measured in the Anaconda area, and little information could be found to support a choice for the dust-loading term. The value for the dust loading parameter during plowing, 0.15 mg/m^3 , was selected primarily on the basis of professional judgement. However, the following analysis was carried out to determine if this choice was in a plausible range for actual dust loading during plowing.

Dust emissions from agricultural plowing, discing, harrowing, etc. may be estimated by:

$$E = k(5.38)s^{0.6} \text{ (Cowherd, et al. 1985)}$$

Where:	E	=	Emissions in kilograms per hectare (kg/ha)
	k	=	Particle size multiplier (unitless)
	s	=	Silt content (percent)

The equation is derived from field testing information and is, therefore, empirical.

An appropriate value for k, 0.21, is provided for particulate size. Silt content may vary considerably; a range of 1.7 to 88% is reported in the literature. A default value of 18% is provided in this reference, without documentation. Without data on silt content of arable soils near Anaconda, this value is used in the analysis.

Assuming that working a hectare would take about 1 hour and that dust emissions would be constant during this time, an emission rate of 69,390 $\mu\text{g/sec}$ is estimated (equivalent to about 0.16 kg/ha). A mixing volume in air for these emissions can be estimated by using a windfield approach. A windfield is calculated by multiplying estimates for wind speed,

horizontal length of emitting surface perpendicular to wind direction, and vertical mixing height. A windfield can be presented in units of m^3/sec , and represents an estimate of the volume of air into which the emitted dust is mixed.

An annual average wind speed of about 9 mph has been estimated for the Anaconda area. Agricultural workers are expected to be in the field frequently during the growing season, and a long term average is appropriate for assessing exposures spanning many days per year for several years.

Emissions during tilling are expected to be caused by mechanical disturbance rather than creation of loose soil which can then be suspended by wind. Thus, the length of the emitting surface is expected to be the length of tractor and plow (or other implement) when the tractor is traveling at right angles to the wind. This length is assumed to be 12 meters (about 40 feet). It is assumed that plowing at different angles to the wind would approximately average out over the course of many days and years and that the above length is plausible.

Assuming a dust load of $150 \mu\text{g}/\text{m}^3$, a vertical mixing height necessary for an emission rate of $69,390 \mu\text{g}/\text{sec}$ is estimated to be about 8.7 meters (approximately 28 feet). This appears to be a reasonable mixing height, suggesting that the assumed dust load of $150 \mu\text{g}/\text{m}^3$ is at least in the plausible range. Therefore, this value is used in calculation of screening levels for the agricultural scenario. The value is used to evaluate exposure during plowing (i.e., 14 days per year).

Uncertainties in the above approach should be noted. The equation provided is sensitive to the silt content of soil. Keeping the windfield constant, dust loading predicted from a silt content of 36% is more than $220 \mu\text{g}/\text{m}^3$, while that for a silt content of 9% is less than $100 \mu\text{g}/\text{m}^3$. Differences in silt content within a factor of 2 from the default value of 18% would, thus, have a significant affect on screening level calculations.

Screening level estimates are also sensitive to windfield assumptions. For example, if the length of the emitting area perpendicular to wind direction is doubled or halved, the resulting dust-loading estimates (18% silt, all other assumptions held constant) are 198 and 49 $\mu\text{g}/\text{m}^3$, respectively.

Screening levels calculated using a dust loading factor of 150 $\mu\text{g}/\text{m}^3$ are expected to be conservative even in light of the above uncertainties. Exposure time, frequency and duration used for screening level calculations can be considered maximum for agricultural workers in the Anaconda region, and most workers would not be exposed as intensely as assumed. Even if average long term dust loading were underestimated by the above analyses, it seems unlikely that actual exposures and risks implied by the screening level estimate will be underestimated.

Exposure Time

Agricultural workers are assumed to work outside for eight hours per day. This value is used to calculate screening levels based on RME and CTE.

6.1.3 COMMERCIAL WORKER SCENARIO

Commercial workers who may become exposed to site contaminants include shopkeepers, office workers, sales people and others that may work at the site. Screening levels for this scenario are based on ingestion of soil and interior dust. Exposure assumptions for the commercial worker scenario are taken from relevant EPA guidance documents and from the Baseline Risk Assessment for the OW/EADA OU (Life Systems 1993). However, different bioavailability factors than were used in Life Systems (1993) are used to calculate screening levels for this HHRA. As discussed above, the BAF_s and BAF_d were derived by EPA (1994a, 1995a) from a recent laboratory study (Battelle 1994).

Exposure assumptions for the commercial worker scenario are shown in Table 6-3 and are discussed below. Screening levels for soil based on this scenario are calculated using the following formulas:

For carcinogens:

$$SL = (TR \times AT \times BW) / (EF \times ED \times IRs \times CFs \times SF_o \times ((FS \times BAF_s) + (C \times FD \times BAF_D)))$$

For noncarcinogens:

$$SL = (TR \times AT \times BW \times RfD) / (EF \times ED \times IRs \times CFs) \times ((FS \times BAF_s) + (C \times FD \times BAF_D))$$

All parameters are described in previous sections.

Averaging Time

Carcinogenic exposures are averaged over 70 years or 25,550 days (EPA 1989a). This value is used to calculate screening levels based on carcinogenic exposure by commercial workers. ATs for noncarcinogenic exposure are 9,125 days for RME and 2,555 days for CTE.

Body Weight

The default BW parameters for adults is 70 kg, respectively (EPA 1989a). This value is used here.

Exposure Frequency

EPA (1993a) presents EFs for evaluation of RME and CTE for commercial workers. These values, 250 and 234 days/year, respectively, are used to calculate screening levels based on the commercial worker scenario.

TABLE 6-3

Exposure Assumptions for the Commercial Worker Scenario

Symbol	Units	Definition	Value	Source
SL	(mg arsenic/kg soil)	risk-based screening level	Section 6-2	-
TR	(unitless)	target risk	Section 6-2	-
AT	(days)	averaging time	Carcinogens = 25,550 Noncarcinogens RME = 9,125 CTE = 2,555	EPA 1989a
BW	(kg)	body weight	70	EPA 1989a
EF	(days/year)	exposure frequency	RME = 250 CTE = 234	EPA 1993a EPA 1993a
ED	(years)	exposure duration	RME = 25 CTE = 7	EPA 1989a Professional Judgement
IRs	(mg/day)	soil ingestion rate	RME = 100 CTE = 50	EPA 1993a EPA 1993a
CFs	(kg/mg)	conversion factor for soil	0.000001	EPA 1989a
SF _O	(mg/kg-day) ⁻¹	oral slope factor for arsenic	1.5	EPA 1995b
BAF _s	(unitless)	bioavailability factor for soil	0.183	EPA 1995a
FS	(unitless)	fraction of soil ingested	0.45	Professional Judgement
C	(unitless)	contribution of soil arsenic to arsenic in dust	0.43	Calculated see text
FD	(unitless)	fraction of dust ingested	0.55	Professional Judgement
BAF _D	(unitless)	bioavailability of interior dust	0.258	EPA 1995a

Exposure Duration

EPA (1989a) recommends using an ED of 25 years to evaluate worker exposures. This value is used to calculate screening levels based on RME to evaluate commercial workers. An ED of seven years is used to calculate screening levels based on CTE. This value is based on professional judgment.

Soil and Dust Ingestion Rate

EPA (1993a) recommends soil and dust IR of 100 mg/day and 50 mg/day for evaluation of RME and CTE, respectively, for workers who may have limited contact with soil. These values are used here.

Fraction of Soil and Dust Ingested

Of the total amount of soil/dust ingested (see Ingestion Rate above), 45% is assumed to be soil and 55% for interior dust.

Bioavailability of Soil and Dust

The BAF_s and BAF_d (18.3% and 25.8%, respectively) were derived by EPA (1994a, 1995a) from data presented in "Determination of Bioavailability of Soluble Arsenic and Arsenic in Soil and Dust Impacted by Smelter Activities Following Oral Administration in Cynomolgus Monkeys" (Battelle 1994).

Contribution of Soil Arsenic to Arsenic in Dust

The contribution of soil arsenic to arsenic in dust was derived from multiple regression analyses performed by Bornschein (1994) on data collected during the Anaconda arsenic exposure study. This is described in detail in Section 3.3.1.2.

6.1.4 RECREATIONAL VISITOR SCENARIO (ADOLESCENT PLAYING IN POOLED WATER)

Adolescents may potentially be exposed to contaminants in surface water, particularly in the surface water adjacent to the Opportunity Ponds. Screening levels for surface water are, therefore, developed for this exposure scenario. Screening levels are based on ingestion of surface water and dermal contact with arsenic in surface water by 4- to 12-year-old children. Exposure assumptions used to calculate screening levels for this exposure scenario are presented in CDM Inc. (1994). Exposure assumptions are summarized in Table 6-4 and are discussed below. Screening levels for this exposure scenario are calculated using the following equations:

For carcinogens:

$$SL = (TR \times AT \times BW) / ((EF \times ED \times SF_O) \times ((IR_{sw} \times CF_{sw} \times ET) + (SA \times PC \times ET \times CF)))$$

For noncarcinogens:

$$SL = (TR \times AT \times BW \times RfD) / ((EF \times ED) \times ((IR_{sw} \times CF_{sw} \times ET) + (SA \times PC \times ET \times EF)))$$

Where:

SL	=	Screening Level for Surface Water (µg/L)
IR _{sw}	=	Surface Water Ingestion Rate (ml/hr)
CF _{sw}	=	Conversion Factor for Surface Water (L/ml)
SA	=	Skin Surface Area Available for Contact (cm ²)
PC	=	Dermal Permeability Constant (cm/hr)
CF	=	Volumetric Conversion Factor (L/cm ³)

All other parameters are defined in previous sections.

Averaging Time

Carcinogenic exposures are averaged over 70 years or 25,550 days (EPA 1989a). This value is used to calculate screening levels based on carcinogenic exposure for the recreational

TABLE 6-4

Exposure Parameters for the Recreational Visitor Scenario

Symbol	Units	Definition	Value	Source
SL	(mg arsenic/L surface water)	risk-based screening level	Section 6-2	-
TR	(unitless)	target risk	Section 6-2	-
AT	(days)	averaging time	Carcinogens = 25,550 Noncarcinogens = 2,920	EPA 1989a
BW	(kg)	body weight	27	EPA 1989b
EF	(days/year)	exposure frequency	RME = 40 CTE = 10	Site-specific Site-specific
ED	(years)	exposure duration	8	Site-specific
IR _{sw}	(ml/hour)	surface water ingestion rate	25	Site-specific
CF _{sw}	(L/ml)	conversion factor	0.001	EPA 1989a
SF _o	(mg/kg-day) ⁻¹	oral slope factor for arsenic	1.5	EPA 1995b
SA	(cm ²)	skin surface area available for contact	10,500	EPA 1989b
PC	(cm/hr)	dermal permeability constant	0.001	EPA 1992a
ET	(hours/day)	exposure time	2	Site-specific
CF	(L/cm ³)	volumetric conversion factor	0.001	EPA 1989a

visitor scenario. AT for noncarcinogens is 2,920 days.

Body Weight

EPA (1989b) presents BW for different age groups of children. These data are used to calculate a representative BW for 4- to 12-year-old children. This value (27 kg) is used here.

Exposure Frequency

Based on the climate in southwestern Montana, exposure to surface water is assumed likely for only five months out of the year. During this time children are assumed to swim or play in the water two times per month and two times per week for evaluation of CTE and RME, respectively. CTE and RME EFs are, therefore, 10 and 40 days per year, respectively.

Exposure Duration

Children from 4-12 years old are evaluated for potential exposure to surface water. The ED for this scenario is therefore eight years.

Surface Water Ingestion Rate

EPA (1989a) suggests that swimmers may ingest 50 mL of surface water per hour. For the exposure scenario evaluated in this HHRA, continuous swimming for 2 hours per day is considered unrealistic. Children are assumed to be playing in the water rather than swimming for most of the 2 hours that they are assumed to be in the water. While some ingestion of water may occur during play, surface water IRs are likely lower during playing activities than during swimming. An IR of 25 mL per hour is therefore considered conservative for this exposure scenario and is used here.

Skin Surface Area Available for Contact

Dermal contact with surface water is expected to occur over the entire body surface. The total body skin surface area for 4- to 12-year-old children is 10,500 cm², respectively. This surface area has been calculated from data for different age groups presented in EPA (1989b).

Dermal Permeability Constant

The dermal permeability constant used here (0.001 centimeters per hour [cm/hr]) is the value presented by EPA (1992a) as a default for inorganic chemicals in aqueous media.

Exposure Time

Exposure time is the number of hours during which recreational visitors are assumed to be swimming or playing in the water. Adolescents are assumed to spend two hours in the water.

6.1.5 RECREATIONAL VISITOR (DIRT BIKER) SCENARIO

Life Systems (1993) report that the Anaconda Smelter NPL Site is used extensively by teenage and adult dirt bikers. Exposure to arsenic in soil is considered possible during dirt biking, and screening values for soil are therefore calculated based on this scenario. Soil screening levels for dirt bike riders are based on ingestion of soil and inhalation of dust. Exposure assumptions for the recreational visitor scenario are taken from Life Systems (1993) and are summarized in Table 6-5 below. The following equation is used to calculate screening levels for this exposure scenario:

$$SL = TR \times AT \times BW / ((EF \times ED) \times ((IRs \times CFs \times SF_o \times BAF_s) + (IR \times SFi \times DL \times ET)))$$

As with agricultural workers, screening levels based on noncarcinogenic exposure cannot be calculated due to the lack of toxicity criteria for evaluation of noncarcinogenic inhalation exposure.

Averaging Time

Carcinogenic exposures are averaged over 70 years or 25,550 days (EPA 1989a). This value is used to calculate screening levels for the recreational dirt biker scenario.

Body Weight

TABLE 6-5

Exposure Assumptions for the Dirt Biker Scenario

Symbol	Units	Definition	Value	Source
SL	(mg arsenic/kg soil)	risk-based screening level	Section 6-2	-
TR	(unitless)	target risk	Section 6-2	-
AT	(days)	averaging time	25550	EPA 1989a
BW	(kg)	body weight	70	EPA 1989a
EF	(days/year)	exposure frequency	RME = 26 CTE = 13	Life Systems 1993 Life Systems 1993
ED	(year)	exposure duration	RME = 30 CTE = 9	EPA 1989a EPA 1989a
IRs	(mg/day)	soil ingestion rate	RME = 100 CTE = 50	Professional Judgement
CFs	(kg/mg)	conversion factor for soil	0.000001	EPA 1989a
SF _o	(mg/kg-day) ⁻¹	oral slope factor for arsenic	1.5	EPA 1995b
BAF _s	(unitless)	bioavailability of soil	0.183	EPA 1995a
IR	(m ³ /hour)	inhalation rate	RME = 2.5 CTE = 1.3	EPA 1989b EPA 1989b
SF _i	(mg/kg-day) ⁻¹	slope factor for inhalation	15	EPA 1995b
DL	(kg/m ³)	dust loading factor	3.8 x 10 ⁻⁷	Professional Judgement
ET	(hours/day)	exposure time	RME = 5 CTE = 2	Lifesystems 1993 Lifesystems 1993

Life Systems (1993) reported that adults represent the majority of people that bike in the Anaconda Smelter Area. The default BW parameters for adults is 70 kg (EPA 1989a). This value is used to calculate screening levels based on the recreational dirt biker scenario.

Exposure Frequency

Life Systems (1993) interviewed trail users at the Anaconda Smelter NPL Site and identified representative EF from these data. Based on this analysis, CTE and RME exposure frequencies of 13 and 26 days per year, respectively, were identified (Life Systems 1993). These values are used to calculate screening levels for the dirt biker scenario.

Exposure Duration

Recreational trail users are assumed to be nearby residents. Residential ED are therefore used for development of screening levels. The RME ED is 30 years, and the CTE ED is nine years. These values represent a 90th percentile and an average estimate for residency at one address (EPA 1989a).

Soil Ingestion Rate

For evaluation of RME and CTE scenarios, dirt bikers are assumed to ingest 100 and 50 mg of soil per day, respectively. These values are based on professional judgement and are selected because a relatively large amount of soil is assumed to be resuspended by trail-bike riding.

Bioavailability of Soil and Dust

The BAF_s of 18.3% was derived by EPA (1994a, 1995a) from data presented in "Determination of Bioavailability of Soluble Arsenic and Arsenic in Soil and Dust Impacted by Smelter Activities Following Oral Administration in Cynomolgus Monkeys" (Battelle 1994).

Inhalation Rate

The BAF_s of 18.3% was derived by EPA (1994a, 1995a) from data presented in "Determination of Bioavailability of Soluble Arsenic and Arsenic in Soil and Dust Impacted by Smelter Activities Following Oral Administration in Cynomolgus Monkeys" (Battelle 1994).

Inhalation Rate

EPA (1989b) presents inhalation rates for adults at moderate and light activity levels. Moderate activity is assumed to evaluate RME and light activity is assumed to evaluate CTE. Inhalation rates for moderate and light activity are 2.5 and 0.8 m³/hour. These rates are used to calculate screening levels for the dirt biker recreational scenario.

Dust Loading Factor

Dust loading during dirt bike riding was estimated using the following approach adopted from Life Systems (1993). A soil emission rate from dirt bike riding is calculated using the following equation based on Cowherd, et al. (1985):

$$E = 0.85 \times (S/10) \times (V/24)^{0.8} \times (W/7)^{0.3} \times (T/6)^{1.2}$$

Where:	E	=	Particulate emission rate (kg/vehicle kilometer traveled (VKT)/hr)
	S	=	Silt content of the soil (%)
	V	=	Vehicle speed (km/hr)
	W	=	Vehicle weight (Mg, where 1 Mg = 1,000 kg)
	T	=	Number of tires (wheels) per vehicle

No adjustment was used to account for days with rain or snow, since this form of the equation calculates emission rates during the dirt-bike riding event (rather than an annual average), and dirt-bike riding is assumed to occur only on dry days. The values of the parameters above were derived as follows:

T The number of tires (wheels) per dirt-bike is two.

Based on these parameters, a value of 0.16 kg/VKT/hr was calculated for E. Multiplying by VKT and dividing by area yields the emission rate in units of kg/hr/m². The value of VKT is given by the number of bikes (assumed to be three) times the speed of each (30 km/hr). Dividing by 3,600 sec/hr results in an estimate of E of 2.0 x 10⁻⁸ kg/sec/m².

The concentration of particulate matter in air resulting from dirt-bike riding at each area were calculated using the estimated soil emission rate and a box model. The following formula was used (Hanna et al. 1982).

$$C = E \times X / (H/2 \times u)$$

Where:

C	=	Concentration of particulate matter in air (kg/m ³)
E	=	Particulate matter emission rate (kg/sec/m ²)
X	=	Distance from upwind to downwind edge of the box (m)
H	=	Mixing height of the box (m)
u	=	Windspeed (m/sec) across the box

Values of these parameters were derived as follows:

- E The emission rate was calculated as described above.
- X The "box" in which riding occurs was assumed to be square. Based on the assumed area of 2E+05 m², this corresponds to a side of about 450 m.
- H The mixing height of the box is a function of distance from the source and turbulence of the air which, in turn, is a function of the roughness of the terrain. The value of H at the upwind edge of the site is assumed to be zero. At the downwind edge, the value of H was calculated from the following equation:

$$X = 6.25Z_0 [(H/Z_0) - 1.58(H/Z_0) + 1.58]$$

Where: X = Upwind to downwind distance (m)
 Z_o = Roughness height (m)

As noted above, X is assumed to be about 450 m. The roughness height is a function of the height of natural and man-made objects (trees, buildings, etc.) in the vicinity of the source. Areas on the Anaconda Site, where dirt-bike riding is assumed to take place, are devoid of buildings and have very few trees. The value of Z_o was, therefore, estimated to be 4 cm (0.04 m), based on the graph presented in Figure 3-6 of Cowherd, et al. (1985).

- u The average wind speed was taken to be 3.6 m/sec, based on annual average values measured at Hiway Junction (MDHES 1988).

Using these input parameters, Life Systems (1993) calculated a dust loading factor of 3.8×10^{-7} kg/m³.

Exposure Time

Based on interviews with recreational trail users at the Anaconda Smelter NPL Site, RME and CTE exposure times of 5 and 2 hours per day, respectively, have been selected as representative. These values are used to calculate screening levels for the dirt biker recreational scenario.

6.2 SCREENING LEVELS FOR THE ANACONDA SMELTER NPL SITE

This section of the HHRA presents screening levels for arsenic based on residential, agricultural and commercial worker and recreational swimmer and dirt biker exposure scenarios. Screening levels for the different exposure scenarios are based on exposure assumptions presented in Section 6.1, and have been developed for a carcinogenic risk range of 10^{-7} to 10^{-3} and a noncarcinogenic HI of 1. Screening levels for the Anaconda Smelter NPL Site are presented in Table 6-6.

TABLE 6-6

**Risk-Based Screening Levels for Arsenic for the Anaconda
Smelter Site**

Medium	Soil								Surface Water	
Screening Level Based on Carcinogenic Risk	Residential Scenario (mg/kg)		Agricultural Scenario (mg/kg)		Commercial Worker Scenario (mg/kg)		Recreational Dirt Biker Scenario (mg/kg)		Recreational Youth/ Swimmer Scenario (mg/L)	
Carcinogenic Risk	RME	CTE	RME	CTE	RME	CTE	RME	CTE	RME	CTE
1×10^{-7}	0.30	1.85	1.00	10.04	1.33	10.15	2.32	53.55	0.002	0.008
1×10^{-6}	2.97	18.5	10.03	100.4	13.3	101.5	23.2	535.5	0.020	0.081
1×10^{-5}	29.7	185.2	100.3	1,003	133	1,015	232.3	5,355	0.20	0.81
1×10^{-4}	297	1,852	1,003	10,038	1,331	10,155	2,323	53,551	2.0	8.1
1×10^{-3}	2,970	18,516	10,033	100,385	13,307	101,546	23,231	535,517	20.2	81.0
Screening Level Based on Noncarcinogenic Effects (HI = 1)	573	1,071	NC	NC	2,139	4,570	NC	NC	1.04	4.16

NC = Not calculated. Risk-based screening levels for these exposure scenarios are based on inhalation and ingestion exposures. A RfC for inhalation is not available; screening levels based on noncarcinogenic effects can, therefore, not be calculated for these exposure scenarios.

7.0 SUMMARY AND CONCLUSIONS

A baseline HHRA was conducted to evaluate the potential human health risks associated with the Anaconda Smelter NPL Site in the absence of remedial (corrective) action. The no action alternative was evaluated in accordance with Sect. 300.430(d) of the NCP. This HHRA focuses on the risks associated with chemicals present in surface soils and groundwater.

7.1 IDENTIFICATION OF CHEMICALS OF POTENTIAL CONCERN

The selection of COPCs for this HHRA is abbreviated due to prior identification of COPCs for OUs of the Anaconda Smelter NPL Site, and for other sites in the upper Clark Fork Basin. COPCs for the remaining areas of the Anaconda Smelter NPL Site are expected to be similar to those for OUs within the site and for sites in the region. Selection of COPCs is limited, therefore, to determination of whether arsenic, cadmium, copper, lead, and zinc should be included in the quantitative assessment. Following a review of the available data, COPCs for the site were determined to be arsenic and lead in soil and arsenic in groundwater.

7.2 EXPOSURE ASSESSMENT

Potential pathways by which humans could be exposed to COPCs at the Anaconda Smelter NPL Site were identified and selected for evaluation. The potential receptors and pathways of exposure selected for evaluation in this HHRA were as follows:

Exposure Pathways for Residents (Adults and Children Ages 0 - 6):

- Ingestion of surface soils
- Ingestion of interior dust
- Ingestion of groundwater

Agricultural Workers (Adults):

- Ingestion of surface soils

- Inhalation of dust

Recreational Users (Dirt Bike Riders):

- Ingestion of surface soils
- Inhalation of dust

Recreational Visitors (Swimmers):

- Ingestion of surface water
- Dermal exposure to surface water

Commercial Workers (Adults):

- Ingestion of surface soils
- Ingestion of interior dust

Data quantity is sufficient to perform a quantitative risk assessment only in the towns of Anaconda and Opportunity. Data quantity is inadequate to quantitatively evaluate risks to receptors throughout the rest of the site. Risk-based screening levels of arsenic in media will be developed for receptors located in areas outside of Anaconda and Opportunity (Section 6.0). These screening levels will be used in conjunction with maps of kriged data to evaluate risks in other areas of the site.

Statistical comparisons of soil data indicate that arsenic concentrations in PTI (1992 and 1993) and Bornschein (1992 and 1994) data are comparable; however, Bornschein (1992 and 1994) data were collected from areas where receptors might have actual exposure. Therefore, only Bornschein (1992 and 1994) soil sampling data are used to evaluate risks. In this study, Anaconda was separated into subareas (A, B, C, D, E, F1, F2, I, and J) to better characterize possible differences in exposure conditions within the community. Opportunity was retained as a separate study (area G). Numerous yards within each subarea were sampled. Soil samples were collected from several locations within each yard, including play, house perimeter, garden, hardpack, and bare areas. Soil concentrations were averaged for each yard.

An exposure point concentration was derived for each area by calculating the 95% UCL of the mean (EPA 1992b) of the arithmetic average soil concentrations for each residence.

Dust data was provided in the Bornschein (1992 and 1994) study. Interior dust samples were collected from homes in Anaconda and Opportunity. Samples were collected from several locations inside each home, and an average concentration for each home was developed.

Statistical tests of dust COPC concentrations demonstrated a lognormal distribution.

Therefore, the data were logtransformed. The exposure point concentration was derived for each area by calculating the upper 95% confidence limit of the arithmetic mean (EPA 1992b) for the lognormalized average interior dust concentrations for each residence.

For Opportunity, both Bornschein (1992 and 1994) and CDM Federal (1994a) groundwater data were used to evaluate risks. Only Bornschein (1992 and 1994) presented groundwater data for the town of Anaconda. Groundwater data were available only for subarea A; all other subareas used public water supply. The exposure point concentration for groundwater is the upper 95% confidence limit of the mean (EPA 1992b) for the groundwater concentrations measured in each subarea.

Arsenic CDI was estimated for each residential exposure pathway based on estimates regarding the extent, frequency, and duration of exposures and the exposure point concentrations. Site-specific exposure assumptions were used when available; these include estimates of arsenic bioavailability in dust, soil, and water. EPA has used available data to derive site-specific arsenic bioavailability estimates for ingested soil and dust (EPA 1994a, 1995a). The following are the bioavailability values used in the HHRA:

- 25.8% bioavailability for dust
- 18.3% bioavailability for soil
- 100% bioavailability for water

Findings in the Anaconda soil ingestion study support the Superfund Program's usual approach of assuming ingestion of 100 mg soil and dust per day as a CTE assumption and 200 mg soil and dust per day as a RME assumption for IRs of children age 0 - 6 years. Though default assumptions are used for soil IR for children, these assumptions are clearly consistent with available site-specific data.

Predictions of exposure obtained from calculations of CDIs were compared to measured exposures of urine arsenic concentrations for children living in Anaconda. The arithmetic and geometric means of predicted and measured urinary arsenic concentrations for these children were compared to evaluate the appropriateness of the exposure assumptions used. Kruskal-Wallis one-way analysis of variance demonstrated that measured and predicted urinary arsenic are not statistically different. However, EPA exposure calculations underpredict urinary arsenic concentrations where measured levels are greater than 10 µg/L. Overall, the results of the comparison support the use of the described exposure calculations in risk assessment for the Anaconda Smelter NPL Site.

7.3 HUMAN RISK CHARACTERIZATION

This section presents the risk characterization based on exposure to arsenic and lead through the pathways selected in Section 3.2. Toxicity values for arsenic (Section 4.1) are combined with CDI to estimate quantitative health risk estimates for exposure to arsenic. Lead toxicity was assessed using the IEUBK Lead Model, Version 0.99. Children (aged 0-6) are considered the sensitive subpopulation at risk for adverse effects due to exposure to lead in environmental media. Risks to adults from lead exposure were not evaluated. A summary of toxicity estimates is presented in Section 5.3.

7.3.1 SUMMARY OF CANCER RISKS

Carcinogenic risks were calculated by multiplying estimates of arsenic CDI by the arsenic-specific oral CSF. The total cancer risks for all pathways for each subarea range from 2.0E-05 to 5.5E-05 for the RME scenario, and from 3.2E-06 to 7.0E-06 for the CTE scenario.

7.3.2 SUMMARY OF NONCARCINOGENIC RISKS

Noncarcinogenic risks were calculated by dividing CDI of arsenic for each pathway by the arsenic-specific oral RfD. The total noncancer risks for all pathways for each subarea range from 0.28 to 0.60 for the RME scenario, and from 0.15 to 0.31 for the CTE scenario. These risks are less than unity, indicating there is little potential for adverse noncarcinogenic effects.

7.3.3 LEAD TOXICITY

EPA generally considers risks from exposure to lead unacceptable if more than 5% of the children have blood-lead levels in excess of 10 µg/dL. Results of the IEUBK modeling indicate that 5% of children in subarea E may have blood-lead levels in excess of 10 µg/dL.

7.4 UNCERTAINTIES

Uncertainties associated with this risk assessment include the following:

- Limited environmental data for areas outside of Anaconda and Opportunity
- Lack of data for lead in interior dust
- Toxicity criteria for arsenic
- Lack of bioavailability data for lead in soils and dust from the study area
- Lack of suitable methodology for evaluating dermal exposure to contaminated soils

- Use of default exposure assumptions and professional judgement in estimating CDI for arsenic and blood-lead levels for lead

8.0 REFERENCES

8.1 GENERAL REFERENCES FOR THE RISK ASSESSMENT

- Agency for Toxic Substances and Disease Registry (ATSDR). 1991a. Toxicological Profile for Arsenic. United States Department of Health and Human Services, Public Health Service. Atlanta: ATSDR.
- Atlantic Richfield Company (ARCO). 1991. Smelter Hill Remedial Investigation and Feasibility Study. Preliminary Site Characterization Information. November.
- Battelle. 1994. Determination of the Bioavailability of Soluble Arsenic and Arsenic in Soil and Dust Impacted by the Smelter Activities Following Oral Administration in Cynomolgus Monkeys. Amended Final Report. March.
- Beck, B.D., P.D. Boardman, G.C. Hook, R.A. Rudel, T.M. Slayton, and H. Carlson-Lynch. 1995. Response to Smith et al. (letter). *Environ Health Perspect.* 103:15-17.
- Bornschein, R. 1991. The Butte-Silver Bow Environmental Health Lead Study. June.
- Bornschein, R. 1992. Anaconda Childhood Arsenic Exposure Study. University of Cincinnati, Ohio. Prepared for Atlantic Richfield Company. April.
- Bornschein, R. 1993. Housing and Ranch Surveys and Family, Environmental, and Census Questionnaires. University of Cincinnati. March 2.
- Bornschein, R. 1994. THE ANACONDA STUDY: An Assessment of Residential Arsenic Exposures Among Children Living in the Vicinity of a Former Copper Smelter. University of Cincinnati, Ohio. UPDATE report dated September 6; UPDATE II dated September 27.
- Buchet, J.P., R. Lauwerys, and H. Roels. 1981. Urinary Excretion of Inorganic Arsenic and Its Metabolites After Repeated Ingestion of Sodium Metaarsenite by Volunteers. *Int Arch Occup Environ Health.* 48:111-118.
- Camp Dresser & McKee Inc. (CDM Inc.). 1985. Assessment of Health Effects Associated with Airborne Transport of Hazardous Substances from the Anaconda Smelter NPL Site. Final Report. Prepared for EPA, Region VIII. November.
- Camp Dresser & McKee Inc. (CDM Inc.). 1994. Baseline Risk Assessment. Streamside Tailings Operable Unit, Silver Bow Creek NPL Site. October 28.

- Carlson-Lynch, H., B.D. Beck, and P.D. Boardman. 1994. Arsenic Risk Assessment. *Environ Health Perspect.* 102:354-356.
- CDM Federal Programs Corporation (CDM Federal). 1995. Evaluation of the Data Collected in the Town of Anaconda.
- CDM Federal Programs Corporation (CDM Federal). 1994a. Domestic Water Sampling In and Near Opportunity, Montana: Data Summary Report. Final Report. Prepared for EPA, Region VIII, by CDM Federal Programs Corporation, Golden, Colorado. April 20.
- CDM Federal Programs Corporation (CDM Federal). 1994b. Phase 1 Screening-Level Ecological Assessment: Anaconda Regional Water and Waste and Anaconda Soils Operable Units. Prepared for EPA, Region VIII, by CDM Federal Programs Corporation, Golden, Colorado. November 21.
- Cowherd C., et al. 1985. Midwest Research Institute. Rapid assessment of exposure to particulate emissions from surface contamination sites. Washington, DC: U.S. Environmental Protection Agency. Office of Research and Development, EPA/600/8-85/002.
- Engel, R.R. and O. Receveur. 1993. Arsenic Ingestion and Internal Cancers: A Review (letter). *Am J Epidemiol.* 138:896-897.
- Griffin, S. 1995. Personal communication with J. Lavelle, CDM Inc. September. S. Griffin, EPA Region VIII.
- Hanna S.R., et al. 1982. Handbook on atmospheric diffusion. Oak Ridge, TN: U.S. Department of Energy, Technical Information Center.
- Life Systems. 1993. Baseline Risk Assessment for the Old Works/East Anaconda Development Area. Prepared for EPA, Region VIII by Life Systems, Inc. April 16.
- Marcus, A.H. 1992. Use of site-specific data in model for lead risk assessment and risk management, In: An Update of Exposure and Effects of lead, B. Beck (Ed), *Fund.Appl.Toxicol.* 18: 10-16.
- Montana Department of Health and Environmental Sciences (MDHES). 1988. Wind roses and frequency tables for five sites in the Anaconda, Montana area. Helena, MT: Department of Health and Environmental Science, Air Quality Bureau. November 14.
- MultiTech. 1987. Silver Bow Creek Remedial Investigation. Draft Final Report. Warm Springs Ponds Investigation. Part 1: Report.

- Mushak, Paul and A.F. Crocetti. 1995. Risk and Revisionism in Arsenic Cancer Risk Assessment (commentary). *Environ Health Perspect.* 103:684-689.
- Peccia & Associates and Lisa Bay Consulting. 1990. Anaconda-Deer Lodge County (ADLC) Comprehensive Master Plan. Prepared for Anaconda-Deer Lodge County Planning Board.
- Peccia & Associates, et al. 1992. ADLC Development Permit System.
- PTI. 1992. Anaconda Soils Investigation Data Summary/Data Validation/Data Useability Report – Final (Phase I). Prepared for Atlantic Richfield Company by PTI Environmental Services. November.
- PTI. 1993. Draft Anaconda Soil Investigation – Phase II Data Summary/Data Validation/Data Useability Report. Prepared for Atlantic Richfield Company by PTI Environmental Services. January.
- RCG/Hagler, Bailly. 1995. State of Montana, Natural Resource Damage Program. Terrestrial Resources Injury Assessment Report: Upper Clark Fork River Basin. Study conducted by RCG/Hagler, Bailly for the State of Montana Natural Resource Damage Program. September.
- Roda, S.M. 1995. The Anaconda Study. An Assessment of Residential Arsenic Exposures Among Children Living in the Vicinity of a Former Copper Smelter. Field and Laboratory Quality Control Results. University of Cincinnati, Ohio. January.
- Taskey, R. D. 1972. "Soil Contamination at Anaconda, Montana: History and Influence on Plant Growth." Master's thesis, University of Montana, Missoula. March 10.
- U.S. Environmental Protection Agency (EPA). 1995a. Review of the Battelle Columbus Report: Determination of the Bioavailability of Soluble Arsenic and Arsenic in Soil and Dust Impacted by Smelter Activities following Oral Administration in Cynomolgus Monkeys. Amended Final Report. March.
- U.S. Environmental Protection Agency (EPA). 1995b. Integrated Risk Information System (IRIS).- Online- Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office. Cincinnati, Ohio.
- U.S. Environmental Protection Agency (EPA). 1995c. Evaluation of the Risk from Lead and Arsenic: Sandy Smelter Site, Sandy Utah. July. Draft Final.

- U.S. Environmental Protection Agency (EPA). 1995d. Endangerment Assessment for Bingham Creek, Utah. Phase III Remediation. July.
- U.S. Environmental Protection Agency (EPA). 1995e. Estimating Site-Specific Exposure to Contaminants in Indoor Dust. Region VIII Superfund Technical Guidance (RA-06). September 1995.
- U.S. Environmental Protection Agency (EPA). 1994a. Review of the Battelle Columbus Report: Determination of the Bioavailability of Soluble Arsenic and Arsenic in Soil and Dust Impacted by Smelter Activities following Oral Administration in Cynomolgus Monkeys. August. Laboratory Project ID #SC930261.
- U.S. Environmental Protection Agency (EPA). 1994b. Guidance Manual for the Integrated Uptake Biokinetic Model for Lead in Children. Office of Solid Waste and Emergency Response. Publication 9285.7-15-1. EPA/540/R-93/081.
- U.S. Environmental Protection Agency (EPA). 1994c. Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities. OSWER Directive #9355.4-12. July.
- U.S. Environmental Protection Agency (EPA). 1994d. Health Effects Assessment Summary Tables (HEAST). Office of Solid Waste and Emergency Response. Annual Update, March.
- U.S. Environmental Protection Agency (EPA). 1994e. Risk-based Concentrations Table. Fourth Quarter 1994. EPA Region III. November.
- U.S. Environmental Protection Agency (EPA). 1993a. Superfund's Standard Default Exposure Factors for the Central Tendency and Reasonable Maximum Exposure. Preliminary Review Draft. May.
- U.S. Environmental Protection Agency (EPA). 1993b. Drinking Water Regulations and Health Advisories. Office of Water, EPA. December.
- U.S. Environmental Protection Agency (EPA). 1993c. Draft Drinking Water Criteria Document on Arsenic. Prepared for Office of Water, EPA, by Life Systems, Inc. March 10.
- U.S. Environmental Protection Agency (EPA). 1992a. Dermal Exposure Assessment: Principles and Applications. EPA/600/8-91-011B.
- U.S. Environmental Protection Agency (EPA). 1992b. Supplemental Guidance to RAGS: Calculating the Concentration Term. Office of Solid Waste and Emergency Response.

Office of Emergency and Remedial Response. Washington, D.C. Publication 9285.7-081.

U.S. Environmental Protection Agency (EPA). 1992c. Water Quality Standards. Federal Register. 57:60911-60923. December 22.

U.S. Environmental Protection Agency (EPA). 1991a. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part B): "Development of Risk-Based Preliminary Remediation Goal. Office of Emergency and Remedial Response, EPA. OSWER Directive 9285.7-01B. December.

U.S. Environmental Protection Agency (EPA). 1991b. Role of the Baseline Risk Assessment in Superfund Remedy Selection Decisions. OSWER Directive #9355.0-30. Office of Solid Waste and Emergency Response.

U.S. Environmental Protection Agency (EPA). 1991c. Human Health Evaluation Manual, Supplemental Guidance: Standard Default Exposure Factors. Office of Solid Waste and Emergency Response, EPA. OSWER Directive 9285.6-03. March.

U.S. Environmental Protection Agency (EPA). 1990. Technical Support Document on Lead. Final Draft. ECAO-UN-757. March.

U.S. Environmental Protection Agency (EPA). 1989a. Risk Assessment Guidance Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. Office of Emergency and Remedial Response, EPA. EPA/540/1-89/002. December.

U.S. Environmental Protection Agency (EPA). 1989b. Exposure Factors Handbook. Office of Health and Environmental Assessment, EPA. EPA/600/8-89/043. July.

U.S. Environmental Protection Agency (EPA). 1988a. Test Methods for Evaluating Solid Waste, Volume 1A: Laboratory Manual, Physical/Chemical Methods.

U.S. Environmental Protection Agency (EPA). 1988b. Special Report on Ingested Inorganic Arsenic. Skin Cancer Nutritional Essentiality. EPA/625-3-87/013. Washington, D.C.

U.S. Environmental Protection Agency (EPA). 1986. East Helena, Montana, Child Lead Study. Summer 1983. Final Report, July.

Warner, M.L., L.E. Moore, M.T. Smith, D.A. Kalman, E. Fanning, and A.H. Smith. 1994. Increased Micronuclei in Exfoliated Bladder Cells of Individuals Who Chronically Ingest Arsenic-Contaminated Water in Nevada. *Cancer Epidemiol Biomarkers Prev.* 3:483-590.

Yost, L., R.A. Schoof, H.R. Guo, P.A. Valberg, B.D. Beck, E. Crecelius, E., and Green, P. Bergstrom. 1994. Recalculation of the Oral Toxicity Values for Arsenic Correcting for Dietary Arsenic Intake. Presented at the Society for Environmental Geochemistry and Health Rocky Mountain Conference, Salt Lake City, Utah. July 18-19.

8.2 REFERENCES FOR THE ARSENIC TOXICOLOGICAL PROFILE

Abernathy, C. O., W. Marcus, C. Chen, H. Gibb, P. White. 1989. Report on Arsenic Work Group Meetings (February 23). Memorandum to P. Cook, Office of Drinking Water, U.S. Environmental Protection Agency, and P. Preuss, Office of Regulatory Support and Scientific Management, U.S. Environmental Protection Agency.

Agency for Toxic Substances and Disease Registry (ATSDR). 1991a. Toxicological Profile for Arsenic. United States Department of Health and Human Services, Public Health Service. Atlanta: ATSDR.

Armstrong, C. W., R. B. Stroube, T. Rubio, et al. 1984. Outbreak of fatal arsenic poisoning caused by contaminated drinking water. Arch. Environ. Health. 39:276-279.

Axelsson, O., E. Dahlgren, C. D. Jasson, et al. 1978. Arsenic exposure and mortality: A case referent study from a Swedish copper smelter. Br. J. Ind. Med. 35:8-15.

Battelle. 1994. Determination of the Bioavailability of Soluble Arsenic and Arsenic in Soil and Dust Impacted by the Smelter Activities Following Oral Administration in Cynomolgus Monkeys. Amended Final Report. March.

Bettley, F. R., J. A. O'Shea. 1975. The absorption of arsenic and its relation to carcinoma. Br. J. Dermatol. 92:563-568.

Brown, C. C., K. C. Chu. 1983a. A new method for the analysis of cohort studies: Implications of the multistage theory of carcinogenesis applied to occupational arsenic exposure. Environ. Health Perspect. 50:293-308.

Brown, C. C., K. C. Chu. 1983b. Approaches to epidemiologic analysis of prospective and retrospective studies: Example of lung cancer and exposure to arsenic. In: Proceedings of the SIMS Conference on Environmental Epidemiology: Risk Assessment, June 28 - July 2, 1982, Alta, Utah.

Brown, C. C., K. C. Chu. 1983c. Implications of the multistage theory of carcinogenesis applied to occupational arsenic exposure. J. Natl. Cancer Inst. 70:455-463.

- Buchet, J. P., R. Lauwerys, H. Roels. 1981. Comparison of the urinary excretion of arsenic metabolites after a single dose of sodium arsenite, monomethyl arsonate, or dimethyl arsonate in man. *Int. Arch. Occup. Environ. Health.* 48:71-79.
- Chen, C. J., M. M. Wu, S. S. Lee, et al. 1988. Atherogenicity and carcinogenicity of high-arsenic artesian well water. Multiple risk factors and related malignant neoplasms of blackfoot disease. *Arteriosclerosis.* 8:452-460.
- Chi, I. C., R. Q. Blackwell. 1968. A controlled retrospective study of blackfoot disease, an endemic peripheral gangrene disease in Taiwan. *Am. J. Epidemiol.* 88:7-24.
- Crecelius, E. A. 1977. Changes in the chemical speciation of arsenic following ingestion by man. *Environ. Health Perspect.* 19:147-150.
- Dunlap, L. G. 1921. Perforations of the nasal septum due to inhalation of arsenous oxide. *J. A. M. A.* 76:568-569.
- Enterline, P. E., G. M. Marsh. 1982. Cancer among workers exposed to arsenic and other substances in a copper smelter. *A. J. Epidemiol.* 116:895-911.
- Glazener, F. S., J. G. Ellis, P. K. Johnson. 1968. Electrocardiographic findings with arsenic poisoning. *Calif. Med.* 109:158-162.
- Goldsmith, S., A. H. From. 1986. Arsenic-induced atypical ventricular tachycardia. *N. Engl. J. Med.* 303:1096-1097.
- Heyman, A., J. B. Pfeiffer, R. W. Willett, et al. 1956. Peripheral neuropathy caused by arsenical intoxication: A study of 41 cases with observations on the effects of BAL (2,3 dimercapto-propanol). *N. Engl. J. Med.* 254:401-409.
- Higgins, I., K. Welch, C. Burchfiel. 1982. Mortality of Anaconda smelter workers in relation to arsenic and other exposures. Dept. of Epidemiology, University of Michigan, Ann Arbor, MI.
- Holland, R. H., M. S. McCall, H. C. Lanz. 1959. A study of inhaled arsenic-74 in man. *Cancer Res.* 19:1154-1156.
- Lagerkvist, B. E. A., H. Linderholm, G. F. Nordberg. 1986. Vasospastic tendency and Raynaud's phenomenon in smelter workers exposed to arsenic. *Environ. Res.* 39:465-474.

- Lagerkvist, B. E. A., H. Linderholm, G. F. Nordberg. 1988. Arsenic and Raynaud's phenomenon: Vasospastic tendency and excretion of arsenic in smelter workers before and after the summer vacation. *Int. Arch. Occup. Environ. Health.* 60:361-364.
- Lee-Feldstein, A. 1983. Arsenic and respiratory cancer in man: Follow-up of an occupational study. In: Lederer W. and R. Fensterheim, eds. *Arsenic: Industrial, Biomedical, and Environmental Perspectives.* New York, N. Y.: Van Nostrand Reinhold, 245-265.
- Little, R. E., G. N. Kay, J. B. Cavender, et al. 1990. Torsade de pointes and T-U wave alterans associated with arsenic poisoning. *P. A. C. E.* 13:164-170.
- Mappes, R. 1977. [Experiments on excretion of arsenic in urine]. (In German). *Int. Arch. Occup. Environ. Health.* 40:267-272.
- Marafante, E., M. Vahter 1987. Solubility, retention, and metabolism of intratracheally and orally administered inorganic arsenic compounds in the hamster. *Environ. Res.* 42:72-82.
- Mizuta, N., M. Mizuta, F. Ito, et al. 1956. An outbreak of acute arsenic poisoning caused by arsenic-contaminated soy sauce (shoyu). A clinical report of 220 cases. *Bull. Yamaguchi Med. Sch.* 4:131-149.
- Morton, W. E., G. A. Caron. 1989. Encephalopathy: An uncommon manifestation of workplace arsenic poisoning? *Am. J. Ind. Med.* 15:1-5.
- Pinto, S. S., C. M. McGill. 1953. Arsenic trioxide exposure in industry. *Ind. Med. Surg.* 22:281-287.
- Rhoads, K., C. L. Sanders. 1985. Lung clearance, translocation, and acute toxicity of arsenic, beryllium, cadmium, cobalt, lead, selenium, vanadium, and ytterbium oxides following deposition in rat lung. *Environ. Res.* 36:359-378.
- Tam, G. K., S. M. Charbonneau, G. Lacroix, et al. 1979. Confirmation of inorganic arsenic and dimethylarsenic acid in urine and plasma of dog by ion-exchange and TLC. *Bull. Environ. Contam. Toxicol.* 21:371-374.
- Tseng, W. P. 1977. Effects and dose-response relationships of skin cancer and blackfoot disease with arsenic. *Environ. Health Perspect.* 19:109-119.
- Tseng, W. P. 1989. Blackfoot disease in Taiwan: A 30-year follow-up study. *Angiology.* 40:547-558.

- Tseng, W. P., H. M. Chu, S. W. How, et al. 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. *J. Natl. Cancer Inst.* 40:453-463.
- U.S. Environmental Protection Agency (EPA). 1995a. Review of the Battelle Columbus Report: Determination of the Bioavailability of Soluble Arsenic and Arsenic in Soil and Dust Impacted by Smelter Activities following Oral Administration in *Cynomolgus Monkeys*. Amended Final Report. March.
- U.S. Environmental Protection Agency (EPA). 1995b. Integrated Risk Information System (IRIS). U.S. Environmental Protection Agency.
- U.S. Environmental Protection Agency (EPA). 1994a. Review of the Battelle Columbus Report: Determination of the Bioavailability of Soluble Arsenic and Arsenic in Soil and Dust Impacted by Smelter Activities following Oral Administration in *Cynomolgus Monkeys*. August. Laboratory Project ID #SC930261.
- U.S. Environmental Protection Agency (EPA). 1994d. Health Effects Assessment Summary Tables (HEAST). Office in Research and Development, Office of Emergency and Remedial Response. OHEA ECAO-CIN-821. March.
- U.S. Environmental Protection Agency (EPA). 1989a. Risk Assessment Guidance Superfund, Volume 1, Human Health Evaluation Manual (Part A). Interim Final. Office of Emergency and Remedial Response, EPA. EPA/540/1-89/002. December.
- U.S. Environmental Protection Agency (EPA). 1988b. Special Report on Ingested Arsenic: Skin Cancer; Nutritional Essentiality. Prepared for the Risk Assessment Forum, U.S. Environmental Protection Agency, Washington, DC. EPA/625/3-87/013.
- U.S. Environmental Protection Agency (EPA). 1984a. Health Effects Assessment for Arsenic. Washington, D. C.: U.S. Environmental Protection Agency, Office of Emergency and Remedial Response. EPA/540/1-86/020.
- U.S. Environmental Protection Agency (EPA). 1984b. Health Assessment Document for Inorganic Arsenic. Final Report. Research Triangle Park, N. C.: U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office; 2-1-3-22, 9-1-9-4. EPA 600/8-83-021F.
- Vahter, M. 1983. Metabolism of arsenic. In: Fowler, B. A., ed. *Biological and Environmental Effects of Arsenic*. New York, N. Y.: Elsevier, 171-198.
- Vahter, M., L. Friberg, B. Rahnster, et al. 1986. Airborne arsenic and urinary excretion of metabolites of inorganic arsenic among smelter workers. *Int. Arch. Occup. Environ. Health.* 57:79-91.

Wall, S. 1980. Survival and mortality pattern among Swedish smelter workers. *Int. J. Epidemiol.* 9:73-87.

8.3 REFERENCES FOR THE LEAD TOXICOLOGICAL PROFILE

Agency for Toxic Substances and Disease Registry (ATSDR). 1991b. Draft Toxicological Profile for Lead. U.S. Department of Health and Human Services, Public Health Service. Atlanta: ATSDR.

Azar, A., H. J. Trochhimowicz, M. E. Maxfield. 1973. Review of lead studies in animals carried out at Haskell Laboratory: Two year feeding study and response to hemorrhage study. In: Barth, D., A. Berlin, R. Engel, et al., eds. *Environmental Health Aspects of Lead: Proceedings, International Symposium, October, 1972, Amsterdam, The Netherlands*. Luxembourg: Commission of the European Communities, 199-210.

Center for Disease Control (CDC). 1991. Preventing Lead Poisoning in Young Children.

Chamberlain, A., C. Heard, M. J. Little, et al. 1978. Investigations into lead for motor vehicles. Harwell, United Kingdom: United Kingdom Atomic Energy Authority. Report No. AERE-9198.

Harlan, W. R. 1988. The relationship of blood-lead levels to blood pressure in the U.S. population. *Environ. Health Perspect.* 78:9-13.

Harley, N. H., T. H. Kneip. 1985. An integrated metabolic model for lead in humans of all ages. Final Report to the U.S. Environmental Protection Agency. Contract No. B44899 with New York University School of Medicine. Department of Environmental Medicine, 1-14.

Koller, L. D., N. I. Kerkvliet, J. H. Exon. 1985. Neoplasia induced in male rats fed lead acetate, ethylurea and sodium nitrile. *Toxicologic. Pathol.* 13:50-57.

Mielke, H., S. Burroughs, R. Wade, et al. 1984. Urban lead in Minnesota: Soil transect results of four cities. *Minnesota Academy of Science.* 50:19-24.

Pirkle J. L., J. Schwartz, J. R. Landis, et al. 1985. The relationship between blood-lead levels and blood pressure and its cardiovascular risk implications. *Am. J. Epidemiol.* 121:246-258.

Pocock, S. J., A. G. Shaper, D. Ashby, et al. 1984. Blood-lead concentration, blood pressure, and renal function. *Br. Med. J.* 289:872-874.

- Pocock, S. J., A. G. Shaper, D. Ashby, et al. 1985. Blood-lead and blood pressure in middle-aged men. In: Lekkas, T. D., ed. International Conference: Heavy Metals in the Environment, Vol. 1, September, Athens, Greece. Edinburgh, United Kingdom: CEP Consultants, LTD, 303-305.
- Rabinowitz, M. B., G. W. Wetherill, J. D. Kopple. 1977. Magnitude of lead intake from respiration by normal man. J. Lab Clin. Med. 90:238-248.
- Shacklette, H. T., J. G. Boerngen. 1984. Element Concentrations in Soils and Other Surficial Materials of the Conterminous United States. U.S. Geological Survey Professional Paper 1270.
- Smith, C. M., H. F. DeLuca, Y. Tanaka, et al. 1981. Effect of lead ingestion on functions of vitamin D and its metabolites. J. Nutr. 111:1321-1329.
- U.S. Environmental Protection Agency (EPA). 1995b. Integrated Risk Information System (IRIS) for Lead.
- U.S. Environmental Protection Agency (EPA). 1994c. Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities. OSWER Directive #9355.4-12. July.
- U.S. Environmental Protection Agency (EPA). 1994d. Health Effects Assessment Summary Tables (HEAST). Office of Research and Development, Office of Emergency and Remedial Response. OHEA ECAO-CIN-821. March.
- U.S. Environmental Protection Agency (EPA). 1992c. Water Quality Standards. Federal Register. 57:60911-60923. December 22.
- U.S. Environmental Protection Agency (EPA). 1991c. Human Health Evaluation Manual, Supplemental Guidance: Standard Default Exposure Factors. Office of Solid Waste and Emergency Response, EPA. OSWER Directive 9285.6-03. March.
- U.S. Environmental Protection Agency (EPA). 1991d. Maximum contaminant level goals and national primary drinking water regulations for lead and copper. Federal Register 56: 26461-26564.
- U.S. Environmental Protection Agency (EPA). 1991e. Guidance Manual for Site-Specific Use of the U.S. Environmental Protection Agency Lead Model. Office of Emergency and Remedial Response, U.S. Environmental Protection Agency.
- U.S. Environmental Protection Agency (EPA). 1989a. Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual (Part A). Office of

Emergency and Remedial Response, Washington D. C., U.S. Environmental Protection Agency. EPA/540/1-89/002.

U.S. Environmental Protection Agency (EPA). 1987. Preliminary review of the carcinogenic potential of lead associated with oral exposure. Prepared by the Office of Health and Environmental Assessment, Carcinogenic Assessment Group, Washington, D. C., for the Office of Drinking Water, Office of Solid Waste, and the Office of Emergency and Remedial Response. OHEA-C-267. Int. Drft.

U.S. Environmental Protection Agency (EPA). 1986a. Guidelines for Carcinogenic Risk Assessment. U.S. Environmental Protection Agency. Federal Register. 51:33992.

U.S. Environmental Protection Agency (EPA). 1986b. Air quality criteria for lead. Research Triangle Park, N. C.: U.S. Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office. EPA 600/8-83-028F.

U.S. Environmental Protection Agency (EPA). 1985. National primary drinking water regulations. Synthetic organic chemicals: Inorganic chemicals and microorganisms. U.S. Environmental Protection Agency. Federal Register. 50:46935-47025.

Ziegler, E. E., B. B. Edwards, R. L. Jensen, et al. 1978. Absorption and retention of lead by infants. *Pediatr. Res.* 12:29-34.

APPENDIX A
DATA EVALUATION

RECEIVED

SUSAN W.

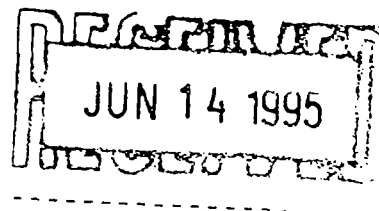


JUN 19 1995

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MEMORANDUM

TO: Bob Alexander, CDM/FPC, Helena
FROM: Dennis Neuman *Dennis Neuman*
DATE: 14 June 1995
SUBJECT: Data Quality

Introduction

The purpose of this Memorandum is to review and comment on the quality of environmental (waters, soils and dusts) and human (urine) data generated by the Kettering Laboratory, Department of Environmental Health, University of Cincinnati Medical Center. This analytical work and subsequent data were in support of the Arsenic Residential Exposure Study conducted in Anaconda, Montana by the Kettering Laboratory. Preliminary review of a document, *Field and Laboratory Quality Control Results* (Roda, S.M., Univ. Of Cincinnati, January 23, 1995), revealed that there were several key pieces of information missing that would prevent such an evaluation. A memorandum (Neuman/MSU to Alexander/CDM-FPC of 24 March 1995) identified information that would allow assessment of the acceptability and useability of the data, and permit calculation of the bias and precision of these data. Ms. Sandy Roda provided (in a Memorandum of 10 May 1995) much of the identified information, and the following text describes the quality of the data to terms of its equivalence to Clark Fork River environmental data produced under CERCLA activities. It was hoped that data would be received that would allow for the quantitative evaluation of data bias and precision. These data are generally not available.

Results of Data Assessment

- * The arsenic water data were judged to be equivalent to Screening Quality Data.
- * The arsenic interior dust/dustfall data were judged to be equivalent to Screening Quality Data.
- * The arsenic soil and exterior dustfall data were judged to be equivalent to Screening Quality Data.
- * The arsenic handwipe data were judged to be equivalent to Screening Quality Data.
- * The arsenic urine data were judged to be equivalent to Screening Quality Data, and concentrations near or below 60 ug/l are most likely biased low.

Detailed Assessments by Matrix

Water

1. Evaluation of written analytical protocols.

Written protocols include the following: For the Perkin Elmer Z-3030 atomic absorption instrument.

- * Analytical standard preparation (10, 25, 50 and 75 ug/l As).
- * Matrix modifier was nickel nitrate.
- * QC samples were made from different source than the calibration standards. Concentration levels were 5 and 10 ug/l.
- * QC sample NIST # 1643C at 82.1 ± 1.2 ug/l was used and diluted to be within the calibration of the instrument.
- * Analytical blank was identified.
- * In every set of 25 samples, there were three QC samples: 5 ug/l, 10 ug/l, and the NIST Standard.
- * Duplicate injections into the ZGFAA were required.
- * Every sample was spiked with 200 ul of 100 ug/l As solution.
- * No separate laboratory duplicate was analyzed.

The protocols for a second instrument, A PE 5100 ZL, was essentially the same, except analytical standards were lower at 5, 10, 20, and 30 ug/l. QC samples were 10 ug/l (from a different source than the calibration standards), and two NIST SRMs. QC samples were run at a frequency of 10%, with the following control limits:

- * QC limits of 17.5 to 21.7 ug/l for 20.5 ug/l standard.
- * QC limits of 27.7 to 33.1 ug/l for 30.0 ug/l standard.
- * QC limits of 10.5 to 14.9 ug/l for 12.5 ug/l continuing calibration standard.
- * Every sample was spiked with As.

2. Evaluation of raw analytical data.

The data set received from Roda was labeled as "dated 1.4.93 (am). As in water." Standards were at 0, 5, 10, 20, 50, 75, and 100 ug/l. The standard curve was acceptable. Sample were analyzed with duplicate injections; coefficients of variation (CV) were acceptable, but somewhat high due to the low absorbance values. QC samples included 5 ug/l SPEX (reported value was 5.2 ug/l), 10 ug/l SPEX (reported value was 10.2 ug/l), and 82.1 ug/l NIST Standard (reported value was 95.2 ug/l). Spike recoveries for the 25 samples in this batch ranged from 93 to 111.3 %. The only analytical deficiency was the lack of analysis of a continuing calibration blank.

3. Evaluation of QC data.

There were approximately 143 water samples analyzed for their As concentration. Each was spiked with As solution. The average recovery \pm std. deviation was 103 ± 6 %, with a range from 88 to 113 %. Results of QC samples were 4.7 ± 0.5 ug/l for the SPEX 5 ug/l standard, 10.0 ± 0.8 for the SPEX 10 ug/l standard, and 81.9 ± 1.8 ug/l for the NIST standard. No laboratory duplicates were analyzed.

4. Evaluation of detection limit information.

The reported detection limit value of 1 ug/l was verified.

5. Summary for As water data.

Based on the analytical protocol, the raw data for the analysis of the first 25 samples, the QC summary, and the recovery of added As, as well as the results for the NIST standard, these water As data are considered to be of acceptable quality. Based on my knowledge of the *Level A/B Criteria*, and the validation protocols established for environmental data at the Clark Fork River NPL Sites, I rate these water As data to be equivalent to *Screening Quality Data*.

Interior Dust and Dustfall Samples

1. Evaluation of written analytical protocols.

The written protocol for the preparation and analysis of interior dust and dustfall samples was very detailed, and included the use of QC samples - reagent blank, method blank, and NIST standards. The digestion technique with 1M and 7M nitric acid should provide complete digestion and recovery of added As. The analytical protocol for As determinations using graphite furnace atomic absorption included the following:

- * Acceptable glassware cleaning method.
- * Analytical standards were 0, 20, 40, 60, 75, and 90 ug/l made from Fisher stock solution.
- * Matrix modifier was nickel nitrate.
- * General protocol was to run set of samples which were diluted 1:10. Samples which required further dilution or concentration were run in a second set.

2. Evaluation of raw analytical data.

The data set received from Rhoda was labeled "Interior dust for As, 10-14-93". Standards were 0, 10, 25, 75, and 90 ug/l, and the standard curve was acceptable. Samples were run with duplicate injections, and the CV values were acceptable. Results for the SPEX 10 and 70 ug/l standards were 9 and 72 ug/l. In the data set provided there were no analysis of preparation, method, or digestion blanks. There was also no analysis of a continuing calibration blank.

3. Evaluation of QC results.

In Roda's memorandum of 10 May 1995, acceptable QC limits were given for blank types including reagent blank, method blank, and the digestion blank. Only one method blank exceeded the QC limit. Acceptable QC limits were also provided for the NIST standards.

Recovery of As added to interior dust samples averaged $101.2 \pm 6.6\%$ (N=58), with a range from 86 to 114 %. Recovery of As added to dustfall samples averaged $105.1 \pm 4.1\%$ (N=7), with a range from 100 - 110 %.

Laboratory duplicate analysis were conducted, but it could not be determined if duplicates were processed through the entire protocol, or if duplicate readings of the same sample were taken at the instrument. The duplicate data revealed excellent precision (generally less than 10% RPD), which remained constant throughout the concentration range.

4. Evaluation of detection limit.

The reported detection limit of 0.002 ug/ml was verified.

5. Summary for interior dust and dust fall samples

Based on the written sample preparation and analytical protocols, raw data, QC results for NIST standards, recovery of added As, and duplicate analysis results, these data are of acceptable quality. Evidence is lacking for determinations of a continuing calibration blank, and in the data set assessed no blanks relating to sample preparation were found. Based on my knowledge of *Level A/B Criteria* and the Clark Fork River Basin protocols for data validation, I judge these data to be equivalent to *Screening Quality Data*.

Soil and Exterior Dust

1. Evaluation of written analytical protocols.

Sampling and drying techniques were acceptable. The cleaning of the 250 um sieve between samples was not rigorous, and it was possible that cross-contamination between samples could have occurred. No preparation blank was used to assess this possibility. The XRF instrumental parameters were described and a 200 second counting time was used. Two standard curves for Pb were prepared from standards obtained from EMSL (Las Vegas) and EPA (Cincinnati). Calibration checks were at 175 and 1040 mg/kg. The instrumental parameters for As were described. Calibration standards were soils from Anaconda that were quantified by atomic absorption after acid digestion. No information on these determinations was provided, nor was any evidence of their As variation presented. These "standards" ranged from 8 to 543 mg/kg. No independent reference material (e.g. NIST SRM) was analyzed to check bias of these As XRF determinations. No duplicate analysis were performed.

2. Evaluation of the raw analytical data.

The data set provided was from 26 August 1993. Counting was 200 seconds and data were reported in ppm.

3. Evaluation of QC results.

The control limits for As check sample were from 17 to 27 mg/kg (low sample), and 254 to 406 mg/kg for the high sample. During the XRF runs the low sample was analyzed 312 times

and results ranged from 17 to 29 mg/kg. The high QC sample was analyzed 311 times with results ranging from 265 to 403 mg/kg. Mean values were near the target levels. There was no evidence of systemic error. No duplicate analysis were performed, as none were stipulated in the analytical protocol

4. Evaluation of the detection limit.

The reported detection limit of 7 mg/kg was verified.

5. Summary fro Soil and Exterior dust samples.

Based on the written protocol, raw data, and QC results these data are judged as acceptable. There were no independent standards (e.g. NIST SRMs) to asses bias of the analytical system. No duplicate samples were analyzed to assess precision. Results of calibration checks were acceptable. Based on the *Level A/B Criteria*, the CFR LAP for XRF determinations, and the CFR data validation protocols, I rate these As data in soil and dust samples equivalent to *Screening Quality Data*.

Handwipe Samples

1. Evaluation of written analytical protocols.

Preparation of handwipe samples using hot acid digestion was appropriate and should provide for complete digestion of the sample, solubilization of As, and good recovery of added As. The protocol described the spiking of clean wipes as control samples which were then treated identically to wipes received from the field. Roda's 10 May 1995 Memorandum states that As was quantified using hydride generation and flame atomic absorption. A separate analytical protocol for this As detection technique was not found in the information received from Roda.

2. Evaluation of raw analytical data.

The data set from Roda was from 3.3.94. Standards were 0, 10, 20, 40, 60, and 80 ug/l. Peak height of the As signal was displayed on a strip chart recorder and then transcribed to the laboratory notebook. After every 10 samples a calibration standard was analyzed, but a calibration blank was not. Every 25 samples a spiked control sample was analyzed and percent recovery determined. In this data set, three spike control samples had recoveries of 110, 95, and 100% recovery. Duplicate analysis were run every 25 samples. It was not possible to determine if the duplicates were prepared prior to sample preparation, or if two readings of the same sample were obtained at the instrument. The analyst completed the data set by rerunning the calibration standards.

3. Evaluation of QC results

Table 4 of Roda's Memorandum shows recovery of As added to the control wipes. The average recovery varied from 85% for the 4 ug spike to 98.7% for wipes spiked with 100 ug. The range of individual recoveries was very broad - from 52.5 to 142%. Results of duplicate analysis were very good, generally less than 10 % RPD. The precision did not vary with concentration.

4. Evaluation of detection limit.

The reported detection limit of 0.1 ug was verified.

5. Summary for handwipe samples.

These As data for handwipes are judged acceptable and to be equivalent to *Screening Quality Data*.

Urine Samples

1. Evaluation of written analytical protocols.

The method presented was based on a literature procedure published in 1981. A mixed standard containing As(III), As(V), methylarsonic acid, and dimethylarsinic acid was prepared and dilutions made to produce working standards. When these standards and/or acidified urine samples were mixed (in a controlled reaction flask) with a solution of NaBH_4 , arsine gas (AsH_3) is formed. This gas is then swept into an absorption tube of an atomic absorption instrument. Only inorganic As in the 3+ valence state will form arsine. Organic forms of As require digestion and As in the 5+ valence state must be reduced to As (III) using a strong reducing reagent, typically potassium iodide, KI. Neither the digestion step or the use of a reducing agent were part of the written protocol.

2. Evaluation of raw analytical data.

The instrument was calibrated with mixed standards ranging from 0 to 75 ug/l. A blank, followed by a spiked urine sample, additional calibration standards, and the NIST standards, were then analyzed. The analytical sequence was then 10 urine samples, calibration standard and blank, etc. A duplicate was analyzed every 20 samples.

3. Evaluation of QC data.

In the data set provided, four NIST standards and three spiked controls were analyzed. Results were 42 and 45 ug/l for the 60 ug/l NIST standard, 460 and 436 ug/l for the 480 ug/l NIST standard, 26 ug/l for the 20 ug/l urine control, and 50 ug/l for the 69 ug/l urine control. In Table 5 of Roda's Memorandum results of the determination of As in the NIST standards is given. For the low standard (60 ug/l As) the mean reported value was 48 ug/l ($N=86$), with a

range of 34 to 61 ug/l. On a percent recovery basis the mean would be 80%, with a range from 56.7 to 101.7%. The high As NIST standard (480 ± 100 ug/l) was analyzed 61 times with an average reported value of 492 ug/l. Reported values ranged from 388 ug/l (80.8% recovery) to 598 ug/l (121.5%). Urine values for As near the 60 ug/l may be biased low. No information on the results of the reference urine samples was provided.

4. Evaluation of the detection limit.

The reported detection limit of 1 ug/l was verified.

5. Summary for urine samples.

Based on the written analytical methods, the raw laboratory data and the results of the analysis of NIST standards, these data are judged as acceptable, with a notation that low level As values (less than 60 ug/l) are mostly likely biased low. These data are equivalent to *Screening Quality Data*.

APPENDIX B

STATISTICAL COMPARISON OF PTI AND BORNSCHEIN SOIL DATA

STATISTICAL COMPARISON OF
BORNSCHEIN ANACONDA SURFACE SOIL ARSENIC DATA
WITH
ANACONDA SOILS INVESTIGATION SURFACE SOIL ARSENIC DATA

This analysis has been performed to resolve two disparate observations made for the Anaconda Superfund Site. First, soil arsenic concentrations for the Anaconda Residential Urinary Arsenic Study (Bornschein, 1992 and 1994) appeared to be less than soil arsenic concentrations found by the Draft Anaconda Soil Investigation Preliminary Site Characterization Report (PSCR) (PTI 1992) for community soils. Second, an ARCO Anaconda representative expressed concern that the Bornschein soil arsenic data would overestimate soil arsenic concentrations in Anaconda because the soil analyzed was a sieved fraction.

This analysis examines the data from both studies to determine (1) if significant differences do exist between arsenic concentrations found in surface soil samples collected for these two studies in Anaconda and nearby communities on an area-by-area basis. And (2) if significant differences do occur, could these differences be explained by differences in sampling methodology. A finding of widespread significant differences would raise the issue of what data set(s) are to be used in the Anaconda Human Health Risk Assessment.

SOILS DATA SOURCES

Arsenic concentrations from results of "Community Soils" were presented in the PSCI Report prepared by PTI Environmental Services for ARCO Anaconda (PTI 1992). Analytical results of the ASI are maintained in the Clark Fork Data Management System by the Clark Fork Data System Manager (CFDSM) for the Department of Health and Environmental Sciences Superfund Section of the State of Montana. Two dBASE files (CDMRISK.DBF and CDMQDV.DBF) containing community, near community, and regional soils analytical data were obtained from the CFDSM. CDM Federal Programs Corporation added additional

information to these files and retained the original file names.

Results of the Anaconda Childhood Arsenic Exposure Study prepared under the direction of Dr. Robert Bornschein of the University of Cincinnati for Anaconda - Deer Lodge County and sponsored by ARCO have not been finalized. However, raw dBASE files containing soils data analytical results (ANAC_S.DBF) and form files necessary to link samples with descriptive data including area location (FM2108.DBF and FM2308.DBF) have been provided and were used for this analysis.

SAMPLING AND ANALYTICAL METHODS

The Community Soils surface soil sampling procedure was described in PTI (1991). On page seven of responses to comments, the following addition was to be made to SOP-39 discussing procedures for surface soil sampling:

The following procedures are designed to be used to collect a surface soil sample (0-2 inches). The procedures listed below may be modified in the field based on field and site conditions after appropriate annotations have been made in the appropriate field log book.

- 1) Locate the site as directed in the appropriate sampling and analysis plan.
- 2) Dig a 12-inch square pit to a depth of approximately 8 inches. If an organic layer is present, this layer will be peeled back.
- 3) A stainless steel bowl will be placed in the pit and a sample collected by scraping the face of the pit from the mineral soil (0-2 inch interval) steel spoon.
- 4) All coarse fragments greater than 0.5 inches will be removed from the bowl. The remaining sample will be disaggregated and homogenized in the collection bowl with a stainless steel spoon.
- 5) In the field laboratory, a sufficient quantity of sample will be saved for measurement of soil slurry pH and conductivity.

- 6) The sample will be sent to a laboratory for analysis per the Anaconda Smelter remedial investigation and feasibility study analytical laboratory protocol (i.e., air dried and sieved to 2mm prior to subsampling for metals).

Surface soil sampling methods for the Bornschein data were described in the first draft of a protocol to study arsenic exposure in children living in or near Anaconda (Bornschein 1992). The following quotes were excerpted from this document. "Soil cores, of 2 cm depth, will be taken in grassy areas and gardens." "A composite sample of soil cores will be taken from grassy yards adjacent to a residence i.e. from the front, back and sides, with 8 to 12 samples per composite. Cores will be taken at approximately equal spacings along the sides of the building, at a distance of one meter (3 feet) from the building wall. Small lot sizes and fences preclude taking building perimeter samples at a distance of one meter on some properties. For large multi-family buildings, proportionately more composite samples will be taken. A composite of soil cores will also be collected from cultivated areas (vegetable and flower gardens) accessible to children, bare areas in yards, obvious play areas and sand boxes. Field duplicates will be collected at 10% of the sample sites." "Soil and dust samples will be air dried overnight. They will then be sieved into two fractions: a 'Large Soil Fraction' which passed a 2mm sieve but not a 250um sieve and a 'Fine Fraction' which passed a 250 um sieve." The Fine Fractions were analyzed by a laboratory based X-ray fluorescence (XRF) unit. Lead (Pb) and arsenic were analyzed in these samples so that spectral Pb interference on arsenic could be corrected (Bornschein, 1994). Samples less than two grams were insufficient for XRF and were consequently analyzed by a Zeiss Graphite Furnace Atomic Absorption Spectrophotometer (ZGFAAS).

DATA SELECTION AND FILE PREPARATION

ASI arsenic data used in this analysis came from data records with the following location acronyms (dBASE field STATION in file CDMRISK.DBF):

CMA = Anaconda residences

CTA1 through CTA6, CTA15, CTA21 = Anaconda targeted residences

CMO = Opportunity residences

CMG = Lost Creek residences, homes on Galen Road

CMF = Fairmont Ranches

Only the composited subsample data results were selected for use in this analysis (designated by a "1" in field SS and no "L" in SAMNO). Replicate data (designated by "A" and "B" in field FREP) were averaged so that no one location could bias the results. Non-detects were used at full value. These data have been validated. Therefore, reported arsenic concentrations were used as presented under the assumption that any modification in the concentration caused by blank contamination (designated by "B" in field ASQUAL) was made by the data validator.

A new field (AREA) was added to the database to incorporate the Bornschein Anaconda Geographic Letter Description according to page seven of Bornschein (1994). The descriptions of area boundaries were plotted on a copy of Figure 6, Community and Community Targeted Sampling Station Locations - Anaconda, from the PSCI report (PTI 1992). Areas A through F were assigned to CMA and CTA samples, accordingly. Area G was assigned to Opportunity Samples. Area H was assigned to Lost Creek Samples. Areas I (Teresa Ann Terrace) and J (Cedar Park Homes) are not considered in this analysis, because no community soils data were collected for these areas. Area K was assigned to Fairmont Ranches data.

The Bornschein soils data were drawn from the dBASE file ANAC_S.DBF and linked to areas A through K via form files FM2108.DBF and FM2308.DBF. Data for perimeter, bare ground, hardpack, garden, and play soil types were available. A new field (SOILAVG) was added that contains the calculated arithmetic average across soil types for each yard. It has already been demonstrated (unpublished descriptive statistical analysis of Bornschein

Anaconda media and urinary arsenic data by CDM Federal Corp 1994) that arsenic concentrations from different soil types may come from different populations, thus bringing into question averaging across soil types within yards. The rationale of averaging across a yard is supported by the assumption, in the absence of behavioral data, that the child spends an equal amount of time in each soil type and receives an equal amount of exposure from each soil type. The quality of the Bornschein data is unknown at this time, because insufficient Quality Assurance/Quality Control information has been provided to date to make such a determination.

STATISTICAL TREATMENT

The ASI data are compared with the Bornschein calculated yard soil averages using multiple box plots and the Mann-Whitney U test. All statistics were performed using STATGRAPHICS PLUS version 6.1 (Manguistics, Inc. 1991).

Multiple box plots are a useful exploratory data analysis technique that summarizes data and makes no distributional assumptions about the data. The scales on all three accompanying figures are the same to make direct comparisons more convenient. Each "box" represents the middle 50 percent of the data concentration values. The lower end of the box is essentially at the 25th percentile or lower quartile and the upper end of the box is at the 75th percentile or upper quartile. The difference between the upper quartile and lower quartile is called the interquartile range. The horizontal line drawn inside the box is the median value (close to the geometric mean for a large number of samples). Vertical lines, called whiskers, extend from each end of the box. The lower whisker is drawn from the first quartile to the smallest data point within 1.5 interquartile ranges from the first quartile. The other whisker is drawn from the third quartile to the largest data point within 1.5 interquartile ranges from the third quartile. Data points beyond the whiskers are outliers and are indicated by "+" on the figures.

The Mann-Whitney U test is analogous to the unpaired t-test. The unpaired t-test compares

two samples to support or refute the hypothesis that the two samples are drawn from the same population. The t-test would be appropriate if the data were normally distributed. Previous statistical analysis of the Bornschein soils data indicated the data are not normally distributed, so using the t-test would be inappropriate. The Mann Whitney U test performs an unpaired t-test on the ranks of the data and makes no distributional assumption. The error level was set at 0.05. If the two-tailed probability of equalling or exceeding the test statistic (Z) is less than 0.05, the hypothesis that both samples come from the same population is rejected. If this probability equals or exceeds 0.05, the hypothesis that both samples come from the same population is accepted. Results for each area comparison follow the boxplot figures. Sample 1 is always the Bornschein data and Sample 2 is always the ASI data.

RESULTS

Boxplots were sandwiched and placed on a light table for a quick area comparison. If the boxes for a given area have much overlap, the samples will likely be from the same population. If the boxes do not overlap the samples will likely be from different populations. The light table comparison was made for the ASI data and the Bornschein calculated soil average data. It appeared that data for Areas A, C, and H could be from different populations.

Average Bornschein soil arsenic concentrations across soil types by yard were greater in seven (A-D, F, H-K) of nine areas compared. ASI soil arsenic averages were greater in areas E and G. Mann-Whitney U tests for all areas only confirmed data for Area H probably came from different populations.

CONCLUSIONS

Results of statistical tests indicate that ASI arsenic data and Bornschein arsenic data are not significantly different for eight of nine areas compared. Bornschein soil arsenic

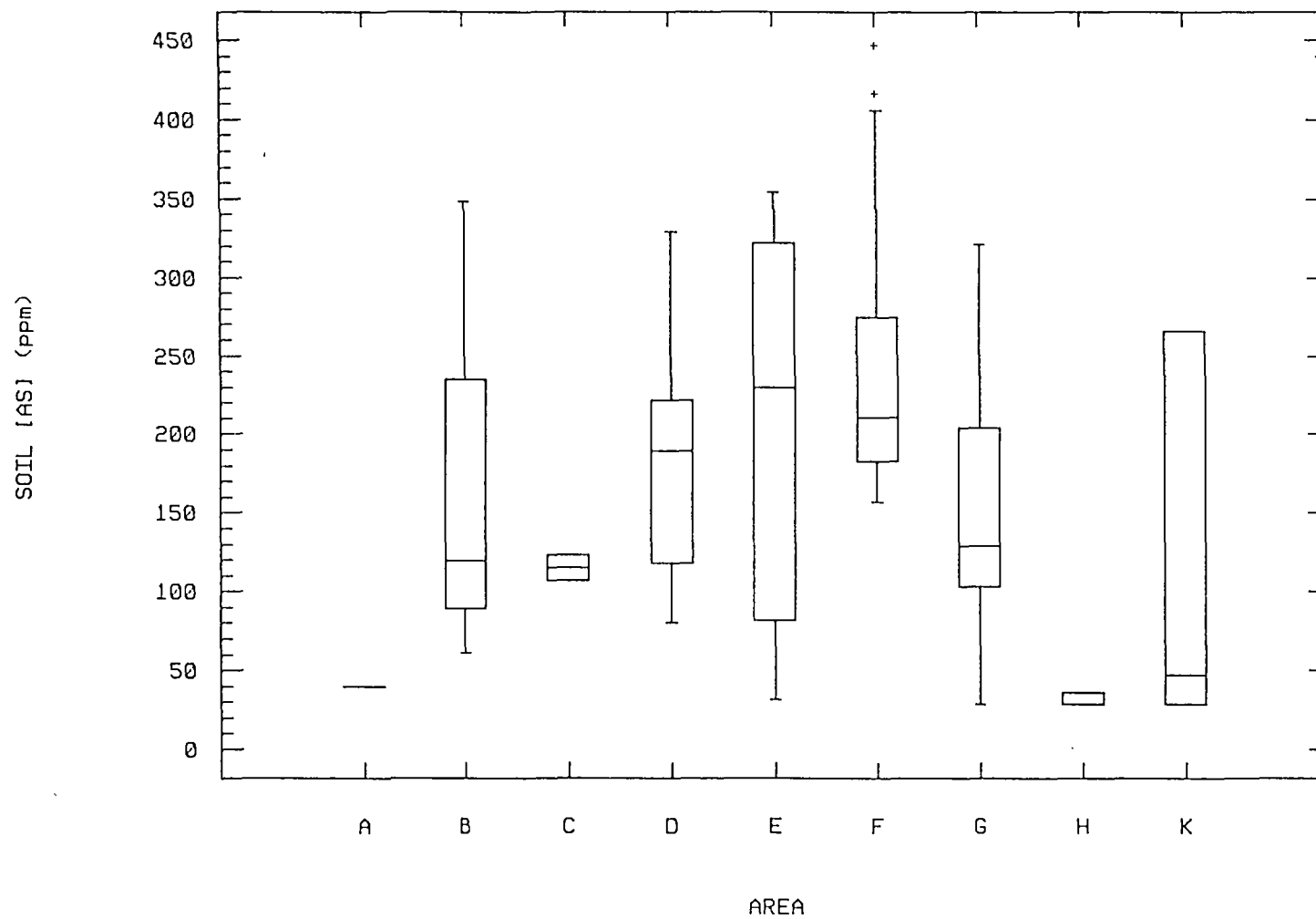
concentrations for Area H were significantly greater (two-tailed $p = 0.05$) than ASI soil arsenic concentrations. These results refute both observations made at the beginning of this analysis, because the two data sets appear to come from one population.

It is suggested that using either data set will result in similar characterization of arsenic in surface soils for Anaconda and nearby communities. Combining the two data sets to maximize sample size appears attractive. If kriging is to be performed, the ASI data have the advantage that sample locations are already known. However, the Bornschein data are about eight times more numerous in Anaconda for Areas A - F than ASI data and more closely approximate the soil fraction to which children are exposed.

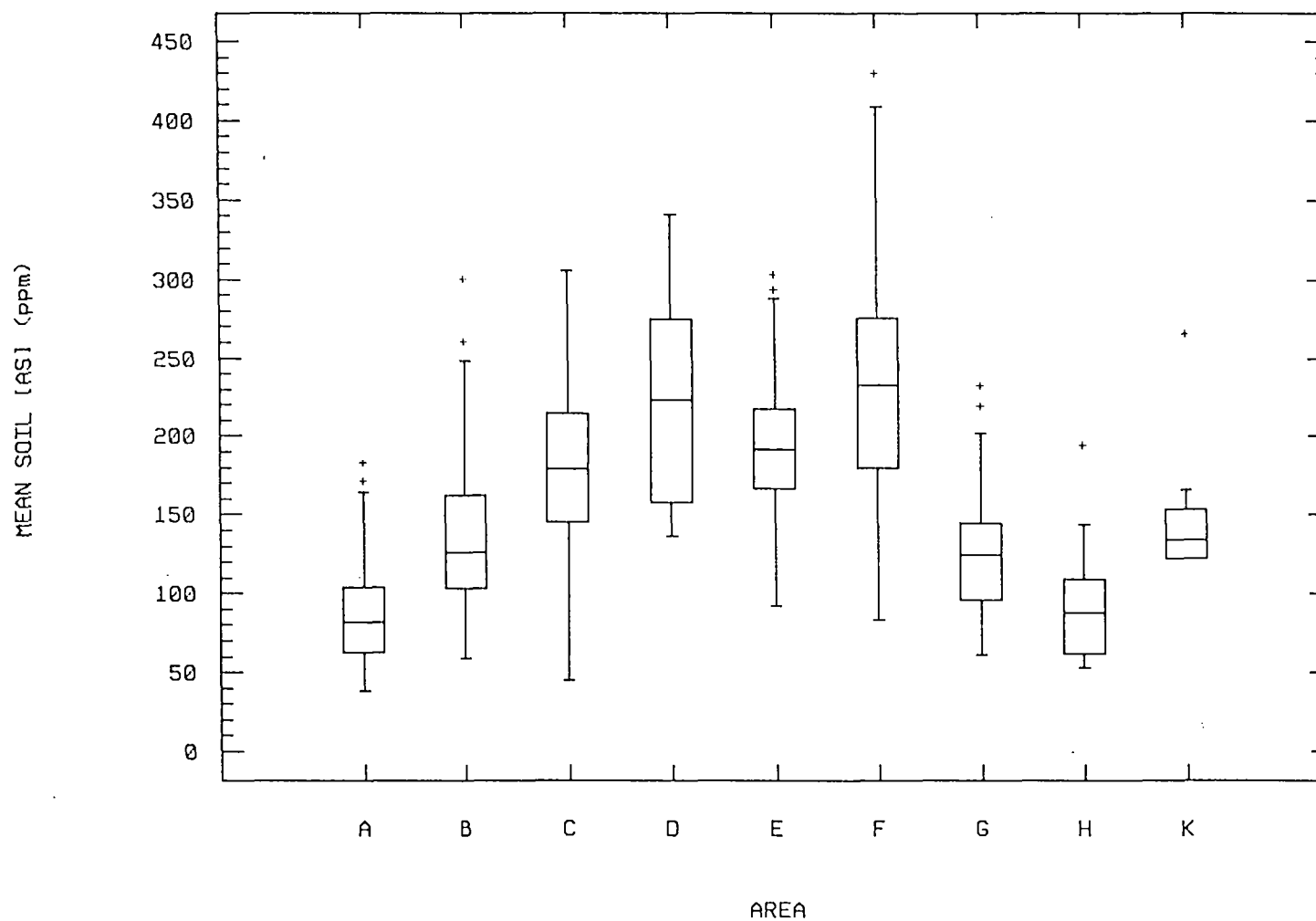
REFERENCES

- Bornschein, R. 1992. Anaconda Childhood Arsenic Exposure Study
Department of Environmental Health. Draft. January 1992. Prepared for Deer
Lodge County. Prepared by University of Cincinnati. Sponsored by Atlantic
Richfield Co.
- Bornschein, R. 1994. The Anaconda Study: An Assessment of Residential Arsenic
Exposures Among Children Living in the Vicinity of a Former Copper Smelter.
UPDATE September 6, 1994. University of Cincinnati.
- PTI. 1991. Draft Sampling and Analysis Plan Anaconda Regional and Community Soil
Investigation. Draft. Prepared by PTI Environmental Services. July 1991.
Prepared for ARCO Anaconda, Montana
- PTI. 1992. Anaconda Soil Investigation Preliminary Site Characterization Information
Report. Draft. Prepared by PTI Environmental Services. March 1992.
Prepared for ARCO Anaconda, Montana. Attached Responses to Comments.

ANACONDA SOILS INVESTIGATION ARSENIC DATA



BORNSCHEIN COMMUNITY SOILS ARSENIC DATA



Comparison of Two Samples

Sample 1: BORN1.SOILAVG SELECT AREA_ID='A'

Sample 2: BORNCOMM.AVGAS SELECT AREA='A'

Test: Unpaired

Average rank of first group = 30.9661 based on 59 values.

Average rank of second group = 3 based on 1 values.

Large sample test statistic Z = -1.55958

Two-tailed probability of equaling or exceeding Z = 0.118858

NOTE: 60 total observations.

Comparison of Two Samples

Sample 1: BORN1.SOILAVG SELECT AREA_ID='B'

Sample 2: BORNCOMM.AVGAS SELECT AREA='B'

Test: Unpaired

Average rank of first group = 37.65 based on 70 values.

Average rank of second group = 34.875 based on 4 values.

Large sample test statistic $Z = -0.239067$

Two-tailed probability of equaling or exceeding $Z = 0.811049$

NOTE: 74 total observations.

Comparison of Two Samples

Sample 1: BORN1.SOILAVG SELECT AREA_ID='C'

Sample 2: BORNCOMM.AVGAS SELECT AREA='C'

Test: Unpaired

Average rank of first group = 20.8243 based on 37 values.

Average rank of second group = 4.75 based on 2 values.

Large sample test statistic $Z = -1.91053$

Two-tailed probability of equaling or exceeding $Z = 0.0560646$

NOTE: 39 total observations.

Comparison of Two Samples

Sample 1: BORN1.SOILAVG SELECT AREA_ID='E'

Sample 2: BORNCOMM.AVGAS SELECT AREA='E'

Test: Unpaired

Average rank of first group = 37.2721 based on 68 values.

Average rank of second group = 40.0833 based on 6 values.

Large sample test statistic Z = 0.297086

Two-tailed probability of equaling or exceeding Z = 0.766397

NOTE: 74 total observations.

Comparison of Two Samples

Sample 1: BORN1.SOILAVG SELECT AREA_ID='D'

Sample 2: BORNCOMM.AVGAS SELECT AREA='D'

Test: Unpaired

Average rank of first group = 20.3261 based on 23 values.

Average rank of second group = 15.2692 based on 13 values.

Large sample test statistic $Z = -1.36688$

Two-tailed probability of equaling or exceeding $Z = 0.171663$

NOTE: 36 total observations.

Comparison of Two Samples

Sample 1: BORN1.SOILAVG SELECT AREA_ID='F'

Sample 2: BORNCOMM.AVGAS SELECT AREA='F'

Test: Unpaired

Average rank of first group = 83.0037 based on 136 values.

Average rank of second group = 82.9828 based on 29 values.

Large sample test statistic $Z = 0$

Two-tailed probability of equaling or exceeding $Z = 1$

NOTE: 165 total observations.

Comparison of Two Samples

Sample 1: BORN1.SOILAVG SELECT AREA_ID='G'

Sample 2: BORNCOMM.AVGAS SELECT AREA='G'

Test: Unpaired

Average rank of first group = 24.4821 based on 28 values.

Average rank of second group = 28.8542 based on 24 values.

Large sample test statistic Z = 1.02809

Two-tailed probability of equaling or exceeding Z = 0.303907

NOTE: 52 total observations.

Comparison of Two Samples

Sample 1: BORN1.SOILAVG SELECT AREA_ID='H'

Sample 2: BORNCOMM.AVGAS SELECT AREA='H'

Test: Unpaired

Average rank of first group = 12 based on 17 values.

Average rank of second group = 2 based on 3 values.

Large sample test statistic Z = -2.64827

Two-tailed probability of equaling or exceeding Z = 0.00809049

NOTE: 20 total observations.

Comparison of Two Samples

Sample 1: BORN1.SOILAVG SELECT AREA_ID='K'

Sample 2: BORNCOMM.AVGAS SELECT AREA='K'

Test: Unpaired

Average rank of first group = 6.5 based on 8 values.

Average rank of second group = 4.66667 based on 3 values.

Large sample test statistic Z = -0.721019

Two-tailed probability of equaling or exceeding Z = 0.470895

NOTE: 11 total observations.

APPENDIX C

DEVELOPMENT OF ARSENIC BIOAVAILABILITY FACTORS



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

REGION VIII

999 18th STREET - SUITE 500
DENVER, COLORADO 80202-2466

NOV 10 1994

MEMORANDUM

SUBJECT: Review of the Battelle Columbus report: ***Determination of the Bioavailability of Soluble Arsenic and Arsenic in Soil and Dust Impacted by Smelter Activities Following Oral Administration in Cynomolgus Monkeys*** (August, 1994) Laboratory Project ID# SC930261.

TO: Charlie Coleman, RPM
Montana Operations Branch

Susan Griffin, Ph.D., D.A.B.T., Toxicologist
Superfund Management Branch

FROM: Christopher Weis, Ph.D., D.A.B.T., Toxicologist
Superfund Management Branch

I have completed a review of the subject report prepared by Battelle for the Atlantic Richfield Company (ARCO) under the direction of Dr. Gary B. Freeman. The work was initiated on November 15, 1993 and completed on August 5, 1994. The purpose of the work was to "***determine and compare the extent of absorption of arsenic in soil and dust impacted from smelter activities near Anaconda, Montana***". The objectives of the work were to: "***determine the extent of absorption and to characterize the rates and routes of excretion of arsenic in Cynomolgus monkeys following a single oral administration of a soluble arsenic solution (sodium arsenate) or a single oral administration (via capsules) of a test soil or dust containing arsenic***." The authors administered a single dose of arsenic (As) as either intravenous, gavage (soluble arsenic) or capsules (soil or dust) to cynomolgus monkeys weighing between 2.42 and 2.88 kg and monitored urinary and fecal excretion of As over a 72 hour period. Absolute percent estimates of arsenic bioavailability were determined by comparing urinary and fecal excretion of arsenic in the IV dose group with urinary and fecal excretion in the oral dose groups.

This work augments earlier work by Battelle aimed at determining the extent of arsenic absorption in lagomorphs. As such, this work adds to the growing database of studies aimed at determining the role of physico-chemical characteristics (concentration, matrix, chemical species, and particle size) in the bioavailability of arsenic. This investigation, if completed and interpreted in juxtaposition with other work in the area of soil/dust exposure characterization for arsenic, might add significantly to the Agency's growing understanding of human exposure to environmental arsenic. The subject work is particularly important as a component of a comprehensive set of data (including an epidemiological investigation conducted by the University of Cincinnati, and a study of childhood soil and dust ingestion conducted by investigators at the University of Massachusetts) collected in the area of Anaconda, Montana during the summer of 1993.



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However, the investigation reviewed herein contains considerable evidence of technical aberration and interpretational inconsistency which may limit its direct application to the stated purpose. Alternative interpretations of the results of the study along with a technical review of the work and suggestions for study completion are presented below.

Comments:

Study Design:

The study was conducted under the general guidelines of Good laboratory Practice (CFR part 792). the work appears to have been generally well planned and carried out with a reasonable amount of care. The introduction to the study presents considerable discussion of a "pilot study" from which the authors draw several conclusions relevant the comparative absorption of arsenic from rabbits and Cynomolgus monkeys; however, no data is presented from the pilot work. Unfortunately, the study design used a limited number of animals and rotated those animals through dose groups as indicated in Table 1. The study design employed animals which had been purchased by Battelle for purposes other than the subject study. The animals employed in the work were received by Battelle on 9/7/90 whereas the study start date was 12/9/93. Therefore the animals had been under observation for considerable time prior to the start of the work. The study design employed three female animals in each of five treatment cycles. The treatment cycles were staggered such that each animal participated in more than one treatment group and such that there were three animals per treatment group at the termination of the study. While certainly an efficient use of resources, several difficulties with the study may have been averted had the investigators employed a full compliment of individual animals to fill the dose groups. The general study design along with treatment groups and animal identifiers is reproduced below:

TABLE 1: Study Design

Treatment Cycle	Date	Animal Number		
		30-537	20-784	30-544
1	12-15-93	Gavage sodium arsenate	Oral Soil (capsules)	IV Sodium Arsenate
2	1-12-94	Oral Soil (capsules)	IV Sodium Arsenate	Oral Dust (capsules)
3	2-2-94	IV Sodium Arsenate	Oral Dust (capsules)	Gavage Sodium Arsenate
4	2-17-94	Oral Dust (capsules)	Gavage Sodium Arsenate	Oral Soil (capsules)
5	3-16-94	—	—	Oral Dust (capsules)

Test Substance Characterization:

Characterization of the **soils** test substance used in the study was completed by PTI of Boulder, Colorado. The test substance was a composite of six play area or bare area soils collected from the 0-2" soil horizon. The samples were dried at 80C then sieved to a particle size of <250 μm . The samples were analyzed for arsenic concentration using XRF then blended to yield a final concentration of 410 ppm. This test substance was analyzed by electron microprobe for mineralogical determination.

House dusts used for dosing were composited samples collected by Hoover Brush vacuum (Model S1137). Samples were collected from carpets in living areas and childrens bedrooms. A decontamination procedure was used between sample collections. Dust samples were then dried at 80C and sieved to < 250 μm .

Frequency of arsenic-bearing particles and mass distributions were calculated. The authors indicate that the averaged results of three "representative" splits of the composite soil sample indicate that the arsenic mineral mass was present primarily as metal-arsenic oxide and iron-arsenic oxide, with lesser contributions from metal-arsenic silicate, enargite, slag, arsenic phosphate and iron-arsenic sulfate. The authors indicate that *"the arsenic mineralogy of the housedust was nearly identical to that of the soil sample"*

Arsenic particle size was <50 μm in diameter with a large number of particles occurring in the < 10 μm fraction. the authors indicate that the house dust particles size was *"slightly larger"* than the soil sample.

The phase association is characterized by the authors as liberated, cemented, and rimmed association, with a high percentage of iron-arsenic oxide phases occurring as liberated particles. The authors state that *"the more frequent occurrence of liberated arsenic particles in the house-dust sample than in the soil could explain the higher observed bioavailability in the house dust"*. This statement, made in the test substance characterization report, indicates that the substance characterization was completed **after** the in life phase of the dosing. According to GLP protocol, test substance characterization should have been completed prior to dosing. ***This is an important and potentially serious broach of GLP protocol.***

Dosing Regimen:

The animals were delivered a single dose of arsenic as indicated in the table below following an overnight fast. Feed was made available to the animals at four hours post dosing and water was made available *ad libitum*. Target doses are as indicated below:

In order to deliver the target dose of arsenic indicated in table 2, and assuming the animal body weights were 2.5 kg, the authors administered approximately 3 grams of soil and approximately 3.8 grams of dust to each animal. The amount of material given to the test system is approximately 20 fold greater than the amount material EPA typically assumes a child might be exposed to and approximately 190 fold greater than the assumed adult dose. It is plausible that such high doses may have a negative influence on the estimates of arsenic absorption made by the authors. However, further work is

necessary to determine the relationship between arsenic dose and percent absorption.

TABLE 2: Dosing Regimen

Treatment	Dose Group (mg As/kg BW)	Number of animals
IV sodium arsenate	0.62 [†]	3
Gavage sodium arsenate	0.62	3
Oral test soil	0.62 [†]	3
Oral test dust	0.26 [‡]	3

The animal weights were approximately 2.5 kg. Therefore the single dose delivered was approximately 1,240 ug of As.

[†] arsenic concentration in soil was 410 ppm.

[‡] arsenic concentration in dust was 170 ppm.

Data collection:

Urine, fecal and cage rinse samples were collected prior to dosing and every 24 hour period for 168 hours (7 days). Assuming that the animals were not chronically dehydrated and that glomerular filtration rate remained stable, such collection protocol should negate the need to normalize urinary arsenic concentrations to creatinine excretion. **Recovery of urinary and fecal arsenic from the IV dose groups was consistently lower than for the other groups (Figure 1).** A one way analysis of variance (ANOVA)

of the means shows that there is a significant difference ($P=0.019$) between the IV group and the oral groups. The authors have normalized the results of urinary recovery from other dose groups to compensate for the poor recovery from the IV group. This correction for poor recovery significantly effects downward the estimates of absorption made by the authors. The observation that recovery of arsenic in the IV group was below that of the oral dose groups does not, by itself, justify the adjustment in absorption calculations which have been made by the authors. No attempt has been made to provide a physiological explanation for the poor recovery observed in the IV group. Nor have the authors taken steps to conduct blood arsenic analysis which may shed light on the observed phenomenon even though significant effort was invested to obtain blood samples. Assuming, as the authors clearly indicate, that delivery of the IV doses was successful and considering the accurate dose verification obtained by the authors, it is likely that arsenic was indeed absorbed by the test system. It is likely that the IV dose remained bound to tissue components, cellular blood components or plasma proteins making it more inaccessible to glomerular filtration or biliary excretion than the oral doses.

Percent Recovery of Dose (F+U)

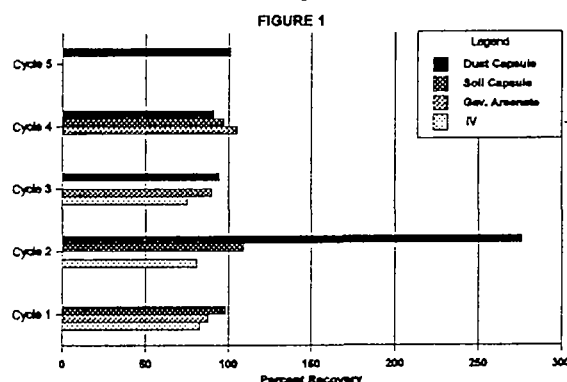
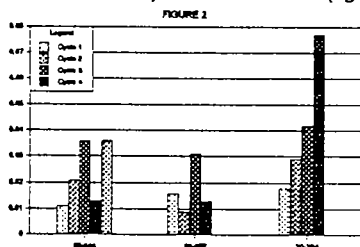


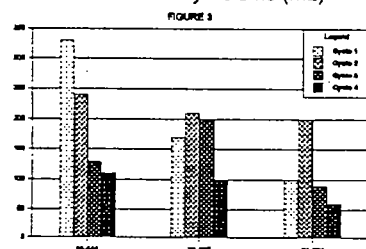
TABLE 3: Percent recovery of Urinary and Fecal Arsenic

Percent Recovery of Urinary and Fecal Arsenic				
	IV Sodium Arsenate	Sodium Arsenate via Gavage	Soil Capsule	Dust Capsule
	82.9	88	98.1	94.4
	81	90.3	109	90.8
	75.3	105	97.2	101
Mean	79.7	94.4	101.4	95.4
Std. Dev.	3.96	9.22	6.57	5.17
SEM	2.28	5.32	3.79	2.99

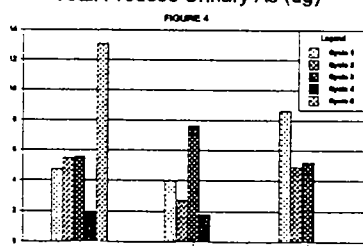
Predose Urinary As Concentration (ug



Predose Urinary Volume (mL)



Total Predose Urinary As (ug)



The authors also collected blood samples at 2,3,4,8,24,48,72,96,120, and 144 hours following oral dose and 2,5,10,15,30,60 minutes and 2,4,8,24,48,72,96, and 120 hours post IV dose (page 21). The blood samples have not been analyzed. **This information is critical to the interpretation and assessment of internal consistency of the Battelle report and the analysis of blood arsenic levels should be completed immediately.** The text of the report (page 23) indicates that the IV doses were delivered slowly over a period of 2-3 minutes which means that the investigators were both delivering the IV dose and collecting the first blood sample at the same time. Unfortunately, this practice is likely to have rendered the first 2 time points unusable for quantitative pharmacokinetics.

The authors indicate that predose urinary arsenic **concentrations** rose throughout the course of the study (Figure 2). They attribute this rise in predose urinary arsenic concentration to an endogenous arsenic source and "correct" for each animal's background arsenic level which had been determined prior to the treatment cycle in question (page 33). However, this interpretation and subsequent data correction appears to be incorrect. Urinary arsenic concentration is increasing due to a concurrent decrease in urinary volume for the predose collection periods and not due to any endogenous arsenic source (Figure 3). Acquisition of concurrent measurements of urinary creatinine may have resolved this question more clearly. In fact, total urinary excretion of arsenic is unchanged during

the predose collection and even appears to decrease somewhat over the course of the dosing cycles (Figure 4). Total urinary arsenic measured at predose is not statistically

different as measured by one way ANOVA. Since the estimate of arsenic bioavailability presented in the report is based upon **total arsenic** excreted ([As] in urine x urine volume), it follows that predose correction for background arsenic should be based upon total arsenic rather than **arsenic concentration**. Table 4 (below) presents summary data on total predose arsenic.

TABLE 4 Summary Statistics for Total Predose Arsenic (μg)

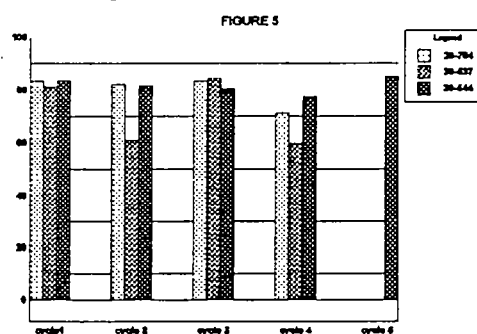
	Animal Number			
	30-544	30-537	20-784	All
Mean	6.18	4.06	6.25	5.49
Std. Dev	4.13	2.56	2.04	3.14
SEM	1.85	1.28	1.18	0.91
Max	13.1	7.64	8.60	13.1
Min	2	1.18	4.49	1.81
Med	5.49	3.39	5.22	5.08

Correction of predose arsenic using total arsenic excreted in urine averaged across animals and cycles would provide a more defensible approach to background correction. As indicated in table 4, the mean arsenic background across the study is 5.49 μg total urinary arsenic.

Food Consumption:

Food consumption for animal number 30-537 was irregular as evidenced in the figure 5. Following each gavage dosing, animal number 30-537's food intake decreased dramatically. The authors indicate that no clinical signs of toxicity were evident and that, perhaps the animal experienced some nausea as a result of the dosing procedure or as a result of the soil or dust dose. The animal's weight remained stable throughout the washout period.

Average Food Consumption (grms)



Clinical Observations:

The authors state that "*clinical observations were made frequently immediately following dosing, the afternoon of the day of dosing, and each day prior to study termination*". However, **no data is presented to substantiate the finding that "[t]here were no abnormal behavioral or clinical signs of toxicity observed in any of the animals during the in-life period of [the] study"**. Given the substantial investment of effort in the study, I would have expected the authors to regularly assess the clinical health of the

animals through; 1) analysis of complete blood counts (CBCs), 2) standard measurements of serum chemistry; 3) assessment of possible disturbance of creatinine metabolism, 4) regular monitoring of body temperature of the animals, and 5) specific observations related to digestive disorders associated with Keflin administration. Perhaps more importantly to the interpretation of study results is the likelihood of systemic staphylococcus or streptococcus infection as a result of surgical implantation of the indwelling catheters used for vascular access. The catheters were implanted on November 19, 1993 whereas the study did not begin until December 9, 1993. The animals were treated with Keflin (a broad-spectrum cephalosporin antibiotic) twice daily indicating that the investigators recognized the possibility of bacteriological infection associated with catheter implantation. One animal (20-784) received treatments of urokinase to re-establish patency of the catheter indicating the possibility of hematoma associated with the access port.

The absence of adequate clinical data and veterinary observations of the animals within the study report is a serious oversight which limits the interpretation of the results. This data may, however, be available within the laboratory notebooks associated with the study and should be requested from the authors.

Study Interpretation:

The authors present graphical results of their estimates of arsenic bioavailability in terms of total excretory arsenic in figures 1 and 2 of the report. Evidence is presented which supports the hypothesis that arsenic in soils is less bioavailable to the test system than soluble IV or soluble oral arsenic. Estimates of absorption of soluble arsenic delivered orally are more or less consistent with literature values available for these forms of arsenic. Due to aberrations in study implementation, two correction methods were used by the authors to estimate bioavailability which were, in my opinion, inappropriate. First, the authors argue that, since the recovery of the IV dose was significantly less than the oral doses, a correction should be made to account for the discrepancy. Second, the authors argue that the predose urinary arsenic concentrations increase throughout the study and consequently should be corrected for endogenous arsenic. Neither adjustment is justifiable based upon the data presented in the Battelle report.

Recommendations:

This investigation could plausibly provide important insight into the physiology and pharmacokinetics of arsenic absorption. With some exceptions, the study is well designed and appears to have been carefully conducted. Additionally, if completed, the investigation may provide a basis for site specific adjustments in arsenic bioavailability for the Anaconda NPL site. However, in its present state, I recommend that the study be rejected as a tool for site specific decision-making. Steps to be taken which might improve the usefulness of the study include: (1) complete analysis of all archived blood samples and thorough comparison of these analysis with data on urinary and fecal excretion in an attempt to resolve the enigma of poor IV dose recovery; (2) provision of adequate information on the clinical chemistry and health of the animals beyond an assessment of arsenic toxicity to include CBCs if available, and clinical observations of the attending veterinarian; (3) reassessment of arsenic absorption without the assumption of an increasing predose endogenous arsenic source. The background arsenic should be

treated as stable and bioavailability estimates presented as corrected for study mean arsenic background; and (4) estimates of arsenic bioavailability should be made assuming that the intravenous dose was delivered as was clearly indicated by the investigators and that it was fully absorbed and physiologically unrecoverable. This approach would be generally more supportable than assuming, as the authors have, that some portion of the IV arsenic was lost in the analysis.

Thank you for the opportunity to review this important and interesting study. I look forward to reviewing it again upon its completion. If you have any questions, please call me at (303) 294-7566.



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JUL 3 1995

MEMORANDUM

SUBJECT: Addendum to the Review of the Battelle Columbus report:
***Determination of the Bioavailability of Soluble Arsenic and
Arsenic in Soil and Dust Impacted By Smelter Activities
Following Oral Administration in Cynomolgus Monkeys.
Amended Final Report (March 1995)***

TO: Charlie Coleman, RPM
Montana Operations Branch

Susan Griffin, Ph.D., D.A.B.T., Toxicologist
Superfund Management Branch

FROM: Christopher Weis, Ph.D., D.A.B.T., Toxicologist
Superfund Management Branch

After review of the initial report (December 1994) EPA requested that the archived blood samples be analyzed and compared to the urinary and fecal excretion data. This memorandum, prepared with technical assistance from Dr. Susan Walker of CDM Federal Programs, Inc., presents the review of the amended subject report prepared by Battelle for the Atlantic Richfield Company (ARCO) under the direction of Dr. Gary B. Freeman.

Study design, test substance characterization, dosing regimen, data collection, food consumption and clinical observations were discussed in the review of the initial report and will not be repeated here.

Analysis of Blood Arsenic Data:

The percent recovery from both matrix spiked samples and the solvent samples was within a technically acceptable range of recovery. Percent recovery in samples of blood spiked with arsenic ranged from 133 percent in the blood samples spiked with 0.02 μg arsenic/sample to 83 percent in the blood samples spiked with 5.00 μg arsenic/sample. The resulting percent residual errors were 20.5 to -3.0 (Figure 1, blood). Percent recovery in samples of solvent spiked with arsenic ranged from 85 percent in the solvent samples spiked with 0.02 μg arsenic/sample to 103 percent in the solvent samples spiked with 5.00 μg arsenic/sample. The resulting percent residual errors were -14.2 to 3.4 (Figure 2). Accuracy of the blood samples was within the technically acceptable range at 0.05 μg /sample. Precision of the study, as measured by the reproducibility of analytical results for the same sample, was not reported.

Absolute bioavailability, measured by evaluating area under the curve (AUC) was



estimated using the trapezoidal method and using AutoCAD. The formula used for the trapezoidal method was as follows:

$$\int_{t_0}^{t_n} \phi(t) dt = \int_{t_0}^{t_1} \phi(t) dt + \dots + \int_{t_{n-1}}^{t_n} \phi(t) dt$$

Where:

$\phi(t)$ is linear between two consecutive blood level-time points, and n is the number of trapezoids into which the curve is divided. Both sets of results were compared to the results reported by Battelle using Sigma Plot to develop estimates (Table 1).

Absolute bioavailability, as estimated using AUC for gavaged dosing with sodium arsenate ranged from 0.67 to 1.07 using Sigma Plot, 0.73 to 1.32 using AutoCAD and 0.9 to 1.22 using the trapezoidal method. These data suggest that the mean absolute bioavailability for gavaged arsenic approaches 100 percent. High absorption efficiencies for gavaged arsenic may be expected and are generally supported in the available literature. The equation used to determine absolute bioavailability was:

$$\frac{\text{AUC for gavage treatment}}{\text{AUC for intravenous treatment}} \times \frac{\text{Total administered dose for intravenous treatment (mg/kg)}}{\text{Total administered dose for gavage treatment (mg/kg)}} \times 100$$

Soil and dust absorption demonstrated a higher variability between monkeys. The range of absolute bioavailability for arsenic in ingested soils among individual animals was from 0.05 to 0.42 resulting in absolute mean bioavailabilities of from 11 to 18 percent. The range of absolute bioavailability for arsenic in ingested dusts were from 0.04 to 0.18 resulting in absolute mean bioavailabilities from 8 to 11 percent.

Monkey 30-537 demonstrated higher absorption of arsenic from ingested soil than did either other monkey when using blood AUC to determine absorption. The 72 hour blood sample from this monkey was extremely high and resulted in an aberrant arsenic absolute bioavailability for ingested soil. According to Dr. Freeman, the sample was re-analyzed with the same analytical results, indicating that the result was not likely to be due to an analytical error. Curve averaging or smoothing were used to limit the impact of the aberrant arsenic value. A second method attempted was to truncate all AUCs at 72 hours. While this had no effect on the intravenous AUC, all other AUCs decreased dramatically. Mean absolute bioavailability estimates decreased from 99 percent to 22 percent for arsenic absorbed from gavage; 22 percent to 4 percent for arsenic absorbed from soil, and 11 percent to 1 percent for arsenic absorbed from dust when curves were truncated, suggesting that bioavailabilities were grossly underestimated using this method (Table 2).

Comparison of mean bioavailability estimates:

As was discussed in the first review, the authors normalized the results of urinary arsenic recovery from other dose groups to compensate for the poor recovery from the IV group. This correction for poor urinary arsenic recovery significantly reduced the estimates of arsenic absorption. Table 3 presents a comparison of the mean absolute bioavailability estimated for urinary arsenic recoveries with and without normalizing the data for poor urinary arsenic recovery. Comparison of the bioavailabilities for gavage, soil ingestion, and dust ingestion developed from the blood arsenic data and the urinary arsenic data demonstrate that the mean absolute bioavailabilities are more comparable (particularly for gavage) when the urinary arsenic bioavailabilities are not normalized for poor arsenic recovery for the IV group (Table 4).

Summary:

The mean absolute bioavailability estimated from urine arsenic concentrations were 91 percent, 18.3 percent and 25.8 percent respectively for gavage, soil and dust (Table 3). The mean absolute bioavailability estimated from blood arsenic concentrations were between 91 and 100 percent for gavage, 11 and 18 percent for soil ingestion and 8 and 11 percent for dust. This study demonstrates that the absorption of arsenic from soils and dust is significantly less than absorption of soluble arsenic from water, and should be used to provide site-specific adjustments in arsenic bioavailability for the Anaconda NPL site.

Figure 1
Percent Residual Error for Spiked Blood

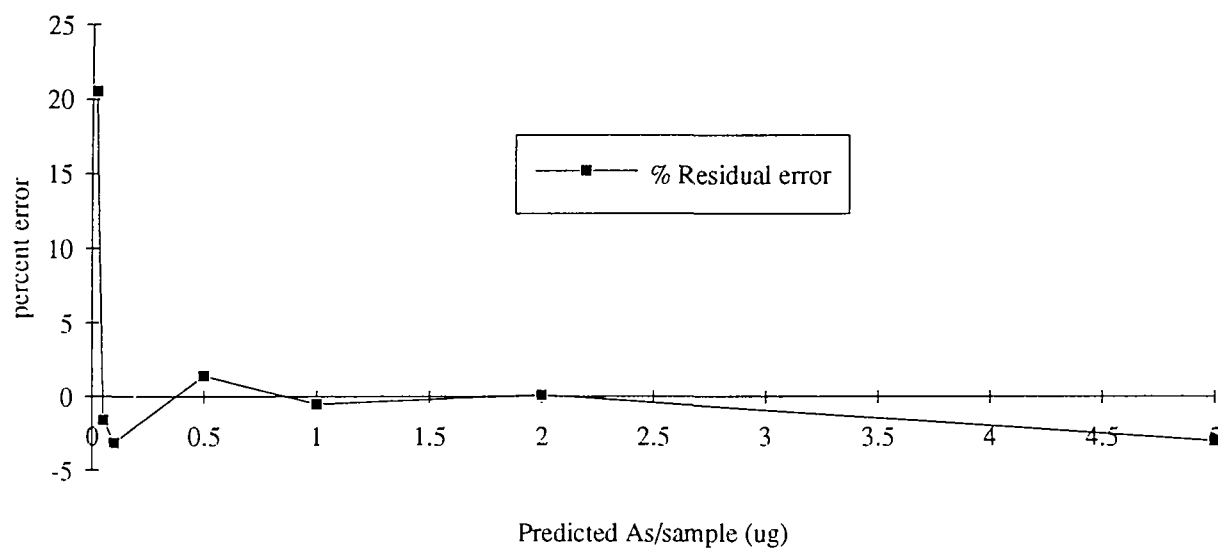


Figure 2
Percent Residual Error for Solvent

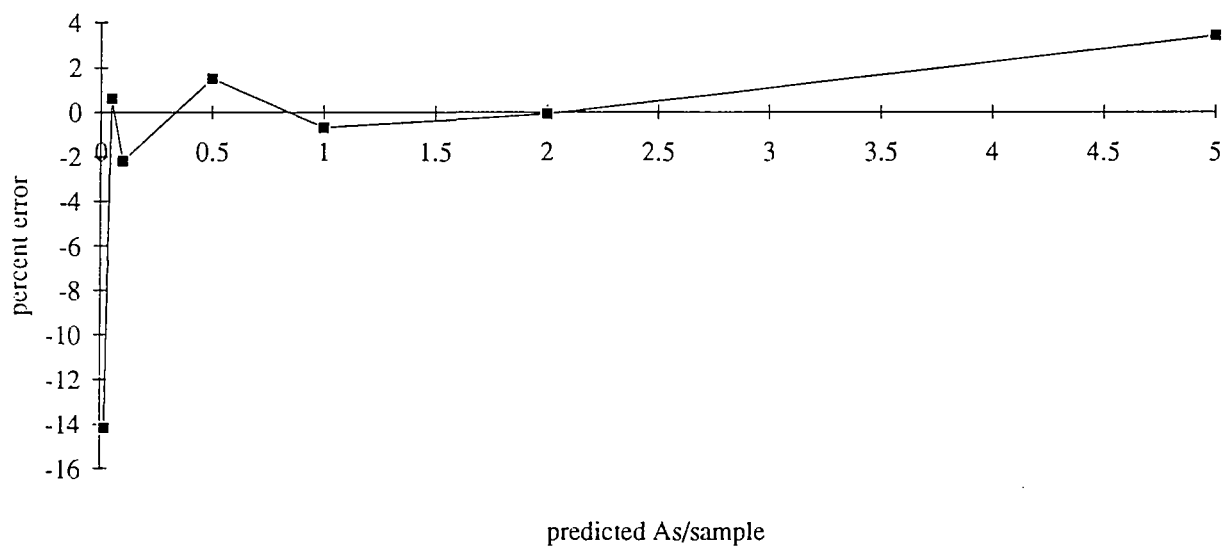


TABLE I
BIOAVAILABILITY ESTIMATED BY AUC MEASUREMENT
BLOOD ARSENIC MEASUREMENTS

Treatment	Monkey	Dose	AREA UNDER THE CURVE					ABSOLUTE BIOAVAILABILITY					MEAN ABSOLUTE BIOAVAILABILITY		
			Uncorrected		corrected			uncorrected		corrected					
			AutoCAD	Trapezoidal	AutoCAD	Trapezoidal	Batelle	AutoCAD	Trapezoidal	AutoCAD	Trapezoidal	Batelle	AutoCAD	Trapezoidal	Batelle
iv	30-544	0.63	4.00	3.80	3.16	3.06	3.18								
iv	20-784	0.60	4.50	3.30	3.90	3.00	4.24								
iv	30-537	0.64	5.20	4.80	3.80	3.75	4.72								
gavage	30-544	0.61	3.69	3.30	3.10	3.10	3.10	0.95	0.90	1.01	1.05	1.01	102%	106%	91%
gavage	20-784	0.60	3.34	3.00	2.83	2.70	2.83	0.74	0.91	0.73	0.90	0.67			
gavage	30-537	0.63	5.31	4.70	4.95	4.50	4.95	1.04	0.99	1.32	1.22	1.07			
soil	30-544	0.58	1.00	0.10	0.20	0.19	0.20	0.27	0.03	0.07	0.07	0.07	15%	18%	11%
soil	20-784	0.62	0.94	0.42	0.20	0.18	0.20	0.20	0.12	0.05	0.06	0.05			
soil*	30-537	0.62	3.46	3.40	1.25	1.54	0.97	0.69	0.73	0.34	0.42	0.21			
dust	30-544	0.26	1.02	0.82	0.24	0.18	0.24	0.62	0.53	0.18	0.14	0.18	11%	8%	10%
dust	20-784	0.26	0.87	0.41	0.08	0.06	0.08	0.45	0.28	0.05	0.05	0.04			
dust	30-537	0.26	0.92	0.51	0.13	0.10	0.13	0.43	0.26	0.09	0.07	0.07			

* = Batelle believed the value for 72 hr blood arsenic in 30-537 to be discrepant. Batelle omitted the value when calculating AUC

AutoCAD used Batelle's corrected numbers to measure area under the curve. Trapezoidal included the 72 hr blood arsenic number

TABLE 2
BIOAVAILABILITY ESTIMATED BY AUC METHOD
TRUNCATED AT 72 HOURS

Treatment	Monkey	Dose	AREA UNDER THE CURVE		ABSOLUTE BIOAVAILABILITY		MEAN ABSOLUTE BIOAVAILABILITY	
			FULL	TRUNCATED	FULL	TRUNCATED	FULL	TRUNCATED
iv	30-544	0.63	183.20	183.20				
iv	20-784	0.60	171.00	171.00				
iv	30-537	0.64	229.12	229.12				
gavage	30-544	0.61	176.16	38.58	0.99	0.22	98.8%	19.6%
gavage	20-784	0.60	152.69	30.32	0.89	0.18		
gavage	30-537	0.63	243.00	43.46	1.08	0.19		
soil	30-544	0.58	3.79	3.79	0.02	0.02	22.4%	3.8%
soil	20-784	0.62	7.28	7.28	0.04	0.04		
soil*	30-537	0.62	135.18	11.34	0.61	0.05		
dust	30-544	0.26	13.83	1.05	0.18	0.01	10.9%	0.8%
dust	20-784	0.26	4.60	0.38	0.06	0.01		
dust	30-537	0.26	7.54	0.46	0.08	0.00		

* = Batelle believed the value for 72 hr blood arsenic in 30-537 to be discrepant. Batelle omitted the value when calculating A AutoCAD used Batelle's corrected numbers to measure area under the curve. Trapezoidal included the 72 hr bloo

TABLE 3
BIOAVAILABILITY ESTIMATED BY AUC MEASUREMENT
URINARY ARSENIC MEASUREMENTS

Treatment	Monkey	ABSOLUTE BIOAVAILABILITY		MEAN ABSOLUTE BIOAVAILABILITY	
		abs	nomal	abs	normalized
iv	30-544				
iv	20-784				
iv	30-537				
gavage	30-544	94%	73%		
gavage	20-784	86%	64%	90.9%	67.6%
gavage	30-537	93%	66%		
soil	30-544	26%	20%		
soil	20-784	16%	12%	18.3%	13.8%
soil*	30-537	13%	9%		
dust	30-544	28%	22%		
dust	20-784	22%	16%	25.8%	19.2%
dust	30-537	28%	20%		

TABLE 4
COMPARISON OF MEAN ABSOLUTE BIOAVAILABILITIES

Treatment	Blood	Urine	Normalized Urine
Gavage	99%	91%	68%
Soil	22%	18%	14%
Dust	11%	26%	19%

APPENDIX D

COMPARISON OF PREDICTED AND MEASURED URINARY ARSENIC

EVALUATION OF THE DATA COLLECTED IN THE TOWN OF ANACONDA USING EPA RISK ASSESSMENT METHODOLOGY

In keeping with EPA risk assessment guidelines, the Exposure Assessment for the Anaconda Smelter NPL site uses factors derived from site-specific data so that risks can be evaluated on a case-by-case basis. Where site-specific data are unavailable, standard EPA default assumptions are used. The following is a discussion of the site-specific data and the EPA default assumptions that will be used to quantify intakes and risks for the residential scenario at the Anaconda Smelter site.

ASSUMPTIONS FOR COMPARISON OF MEASURED TO PREDICTED URINARY ARSENIC LEVELS

Assumptions for Bioavailability of Arsenic from Soils and Dust

Determination of the Bioavailability of Soluble Arsenic and Arsenic in Soil and Dust Impacted By Smelter Activities Following Oral Administration in Cynomolgus Monkeys. Final Report (Battelle December 1994) and Amended Final Report (Battelle March 1995) presented blood arsenic, urine arsenic and feces arsenic data collected from Cynomolgus monkeys exposed to arsenic by intravenous injection, gavage, and capsules containing soil and dust collected at the Anaconda Smelter site. EPA has used these data to derive a site-specific arsenic bioavailability estimates for ingested soil and dust (EPA 1994, 1995)

Bioavailability is defined as the fraction of the amount of arsenic in the system after oral dosing compared to the amount of arsenic in the system after intravenous administration adjusted for the difference in the size of the dose. The mean absolute bioavailability estimated from urine arsenic concentrations were 91 percent, 18.3 percent and 25.8 percent respectively for gavage, soil and dust. The absolute bioavailability estimated from blood arsenic concentrations ranged between 91 and 100 percent for gavage, 11 and 18 percent for soil ingestion and 8 and 11 percent for dust. This study demonstrates that the absorption of arsenic from soils and dust is significantly less than absorption of soluble arsenic from water, and should be used to provide site-specific adjustments in arsenic bioavailability for the Anaconda NPL site. The mean absolute availability estimated from urine arsenic concentrations were used to compare measured and predicted urinary levels in children.

Selection of Exposure Assumptions

This evaluation of the media data (water, soil, dust) collected in Anaconda by Dr. Robert Bornschein using EPA risk assessment methodology serves several purposes. First, the large number of soil and dust samples collected, the data should allow a more accurate analysis of the community than other media data sets. Second, by having measured urinary arsenic, we are able to compare predicted urinary arsenic levels to the measured spot urinary arsenic levels.

The assumptions presented in Table 1 were used to predict excretion of speciated arsenic in ug/L for those children where ages were available (n = 373). The estimates were then graphed

against speciated arsenic for those children where it was measured (n = 366). Total arsenic excretion was also predicted, however, the uncertainty associated with the predicted total arsenic excretion is greater than the uncertainty associated with the predicted speciated arsenic excretion.

It was assumed that all children were in a steady state condition, i.e., the amount of arsenic absorbed (from soil, dust, water, and in the case of total arsenic, food) equals the amount of arsenic excreted in urine.

$$\text{ABSORBED ARSENIC} = \text{EXCRETED ARSENIC}$$

The formula used to estimate daily absorption was as follows:

$$ABS = \frac{C_s \times IR_s \times CF_s \times EF \times BAF_s}{AT} + \frac{C_w \times CF_w \times IR_w \times EF \times BAF_w}{AT}$$

The resulting value (ABS) in mg/day is the estimated absorbed arsenic per day for each individual child. (Refer to the Table below for an explanation of the other terms.)

The following formula was used to estimate the amount of speciated arsenic excreted in the urine:

$$EXC = \frac{ABS \times CF_{abs}}{RATE \times CF_{exc}}$$

Where:

EXC (μg/L) = amount of arsenic excreted in the urine

ABS (mg/day) = The result from the equation above.

CF_{abs} (μg/mg) = Conversion factor for μg/mg = 10³ μg/mg

RATE (ml/day) = The estimated urinary output per day for a given age in months

CF_{exc} (L/ml) = Conversion factor for milliliters to liters = 10⁻³ L/ml

During the Anaconda study, Dr. Bornschein measured the 24-hour urine output of a subset of 25 children. The mean urine volume measured by Dr. Robert Bornschein for each age group is used as the estimated urinary output per day (RATE).

Selection of Ingestion Rate for Children in Living in Anaconda

Dr. Edward Calabrese conducted a soil ingestion study in Anaconda. The study was a week-long measurement of soil ingestion in 64 children. Using a single "best tracer" methodology, the ingestion rate median was 51 mg/day, the mean was 117 mg/day, the 90th percentile was 277 mg/day. The "four best tracers" study resulted in an ingestion rate median of 39 mg/day, a mean of 83 mg/day and a 90th percentile of 273 mg/day. The findings in the Anaconda

soil ingestion study support the Superfund Program's usual approach of 100 mg/day as a central tendency exposure assumption and 200 mg/day as a reasonable maximum exposure assumption for ingestion rates.

Predicting Total Urinary Arsenic Excretion

As a substudy in the soil ingestion study by Edward Calabrese, daily food samples of a subset of 30 children were collected. The mean mg arsenic in food per day was 0.00705 with a standard deviation of 0.0065. To predict total urinary arsenic excretion, it was assumed that children over the age of 18 months ate "solid food". It was also assumed that 100 percent of the arsenic in food was bioavailable and therefore was excreted in the urine. Because of the large standard deviation in the arsenic concentration in food and because of the lack of information on the bioavailability of arsenic in food, there is a large degree of uncertainty associated with predicted total urinary arsenic excretion.

RESULTS OF THE COMPARISON OF PREDICTED SPECIATED URINARY ARSENIC TO MEASURED URINARY ARSENIC IN CHILDREN

Using the assumptions described above, predicted urinary arsenic concentrations were developed for each child. Table 2 presents the measured and predicted speciated and total urinary arsenic levels for each child. The arithmetic and geometric means and standard deviations for measured and predicted urinary arsenic are presented in Table 3.

Kruskal-Wallis one-way analysis of variance demonstrated that the populations from which the measured and predicted data sets were drawn have the same mean. Figure 1 is a comparison of the measured and predicted speciated urinary arsenic and demonstrates that predicted urinary arsenic excretion is similar to the measured urinary arsenic levels. However, the EPA model underpredicts urinary arsenic at measured levels greater than 10 ug/L. Figure 2 compares the measured and predicted total urinary arsenic.

USE OF EPA DEFAULT EQUATION TO PREDICT POTENTIAL CARCINOGENIC RISK

To determine the overall risk for the Anaconda Community Soils data, the soil and dust data from Dr Robert Bornschein's Anaconda Study were evaluated using the following formula:

$$RISK = \frac{C_s \times EF \times ED \times IR \times CF \times BAF \times SF_o}{BW \times AT}$$

C_s (mg/kg) = the concentration of the contaminant in soils and dust.

EF (days/year) = exposure frequency.

ED (years) = exposure duration.

IR (mg/day) = ingestion rate.
CF (kg/mg) = conversion factor of 10^{-6} .
BAF = bioavailability factor
 SF_o (mg/kg/day) $^{-1}$ = the oral slope factor.
BW (kg) = body weight in kg.
AT (days) = Averaging time.

- The dust mean and soil mean were calculated for each area sampled by Dr. Bornschein as well as the entire population.
- The soil concentration was estimated for each yard by calculating the average of all yard soil samples collected.
- The 95% upper confidence limit of the arithmetic mean (ucl of the mean) was calculated for each area as well as the entire population.
- The exposure point concentration (C_s) for each area, and for the entire population assumed 55% dust and 45% soil was ingested. [For the relative bioavailability analysis, the actual ucl of the mean for dust and soil was used.]
- The reasonable maximum exposure ingestion rate for children (0 to 6) (RME) was assumed to be 200 mg/day.
- The central tendency exposure ingestion rate for children (0 to 6) (CTE) was assumed to be 100 mg/day.
- The RME ingestion rate for all others was assumed to be 100 mg/day.
- The CTE ingestion rate for all others was assumed to be 50 mg/day.
- Body weight for children was assumed to be 15 kg.
- Body weight for all others was assumed to be 70 kg.
- Exposure frequency was assumed to be 350 days/year.
- The RME exposure duration was assumed to be 30 years
- The CTE exposure duration was assumed to be 9 years (2 years as a child 0 to 6 and 7 years as an adult)
- Averaging time was assumed to be 365 days/year times 70 years or 25550 days.
- Bioavailability factors were selected from the Cynomolgus monkey study by Battelle for ARCO. The absolute bioavailability selected for dust absorption was 25.8 percent and for soil absorption was 18.3 percent.
- The oral slope factor was 1.75 (mg/kg/day) $^{-1}$.

Table 4 presents the residential risks for Anaconda using the media data collected by Dr. Bornschein and the reasonable maximum exposure assumptions. The range of risks when assuming 25.8 percent bioavailability for arsenic in dust and 18.3 percent bioavailability for arsenic in soils is from 4.9×10^{-5} to 1.2×10^{-4} . The overall community risk would be 7.9×10^{-5} . Area D has the highest overall concentration of arsenic in soil and dust with Area A having the lowest concentration.

Table 5 presents the residential risks for Anaconda using the media data collected by Dr. Bornschein and the central tendency exposure assumptions. The range of risks when assuming 25.8 percent and 18.3 percent bioavailability for soils is from 7.9×10^{-6} to 1.9×10^{-5} . The overall community risk would be 1.3×10^{-5} .

FIGURE 1
MEASURED AND PREDICTED SPECIATED URINARY ARSENIC

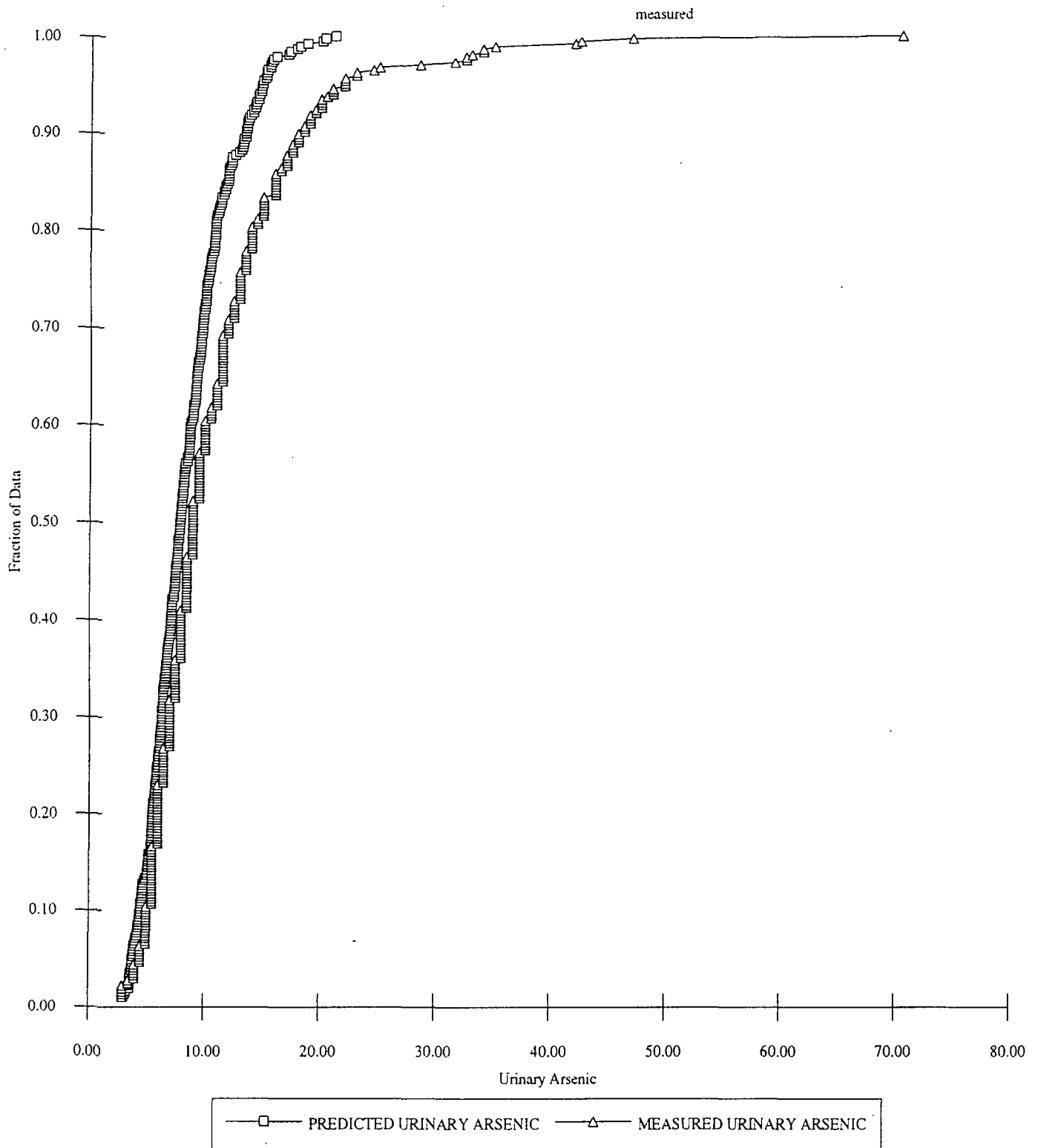


FIGURE 2
COMPARISON OF MEASURED AND PREDICTED TOTAL URINARY ARSENIC

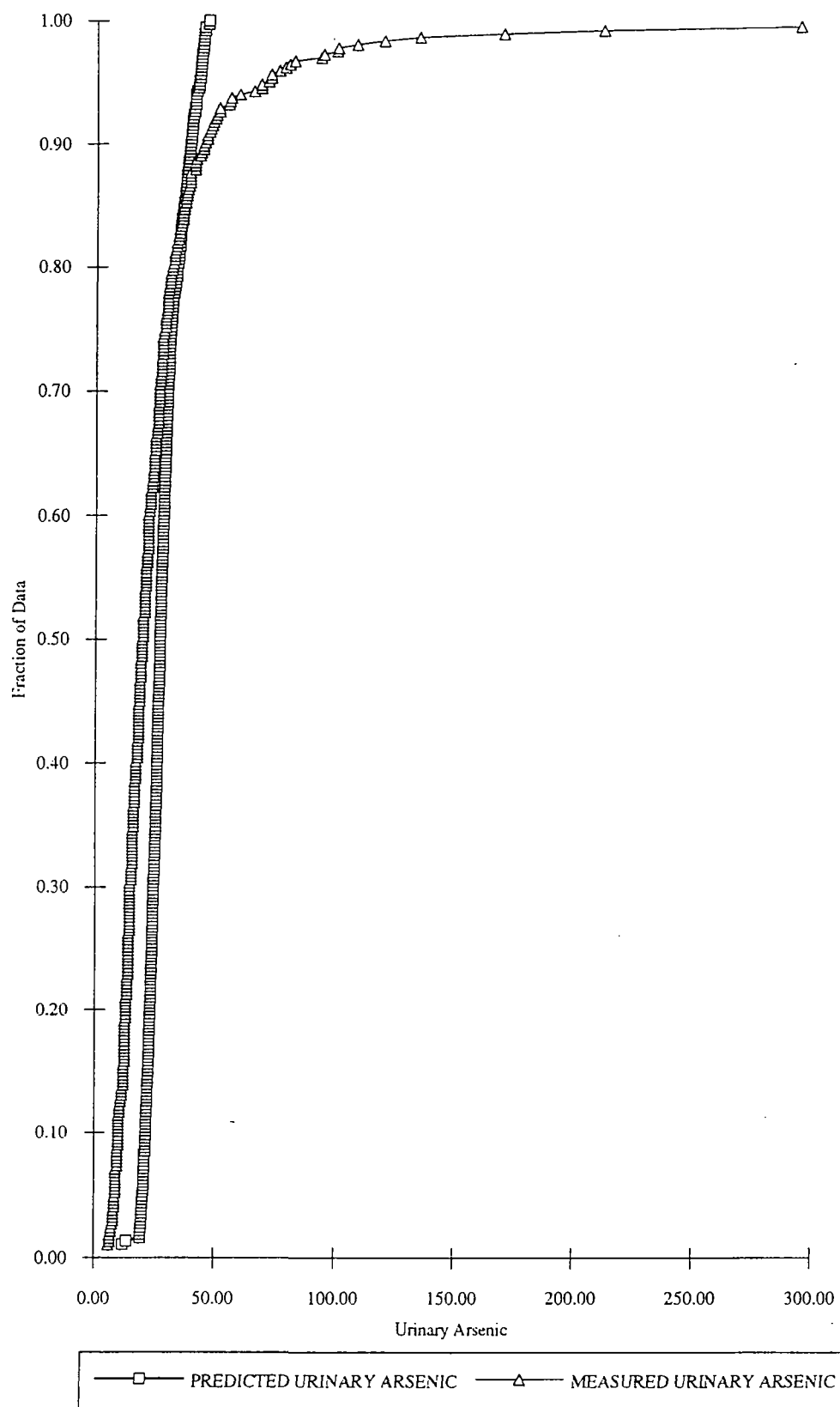


TABLE 1

Symbol	Definition	Value Used
C_s	Arsenic concentration of ingested soil and dust (mg/kg)	55% sampled interior dust As 45% sampled average yard As
IR_s	Ingestion rate of combined soil and dust (mg/day)	100 mg/day
CF_s	Conversion Factor (kg/mg)	10^{-6} kg/mg
EF	Exposure Frequency (days)	350 days
AT	Averaging Time (days)	365 days
C_w	Arsenic concentration of ingested water ($\mu\text{g/L}$)	Measured water arsenic concentration (or 1/2 detection limit - $0.5 \mu\text{g/L}$)
CF_w	Conversion Factor (mg/ μg)	10^{-3} mg/ μg
IR_w	Ingestion Rate (L/day)	0.7 L/day
BAF_s	Bioavailability for soil and dust	25.8 % for dust 18.3 % for soil
BAF_w	Bioavailability for water	100 %
ABS	Estimated absorbed arsenic per day for each individual child (mg/day)	Calculated for each individual child
CF_{abs}	Conversion factor for $\mu\text{g/mg}$	$10^3 \mu\text{g/mg}$
RATE	The estimated urinary output per day for a given age in months (ml/day)	Children less than 36 months of age excrete 240 ml urine/day Children between 36 and 60 months of age excrete 355 ml urine/day Children greater than 60 months of age excrete 432 ml urine/day
CF_{exc}	Conversion factor for milliliters to liters	10^{-3} L/ml

TABLE 2
ACTUAL AND PREDICTED URINARY ARSENIC LEVELS

AGE IN MONTHS	ESTIMATED URINE PRODUCTION	ARSENIC IN WATER (ug/L)	ARSENIC IN DUST (mg/kg)	ARSENIC IN SOIL (mg/kg)	55% DUST 45% SOIL ABSORBED	MEAN SPEC URINARY AS NORMALIZED (ug/L)	NATURAL LOG MEAN SPEC URINARY AS NORMALIZED	MEAN TOTAL URINARY AS NORMALIZED (ug/L)	NATURAL LOG MEAN TOTAL URINARY AS NORMALIZED	EPA PREDICTED SPECIATED AS CTE (ug/L)	NATURAL LOG EPA PREDICTED SPECIATED AS CTE (ug/L)	DIFFERENCE BETWEEN SPECIATED MEASURED AND PREDICTED	EPA PREDICTED TOTAL AS CTE (ug/L)	NATURAL LOG EPA PREDICTED TOTAL AS CTE (ug/L)	DIFFERENCE BETWEEN TOTAL MEASURED AND PREDICTED
8	240	0.5	22.3	154	16	16.00	2.77	35.50	3.57	7.72	2.04	8	7.72	2.04	27.78
14	240	0.5	62.2	266	31	5.00				13.68		(9)	13.68		
15	240	0.5	67.4	206	27	11.50	2.44	23.50	3.16	12.00	2.48	0	12.00	2.48	11.50
20	240	0.5	23.4	181	18					8.69			38.07		
21	240	0.5	23.1	166	17	32.50	3.48	44.50	3.80	8.15	2.10	24	37.53	3.63	6.97
21	240	0.5	72.1	193	26	17.00	2.83	36.00	3.58	11.83	2.47	5	41.20	3.72	(5.20)
23	240	0.5	65.3	246	30	10.00	2.30	10.00	2.30	13.19	2.58	(3)	42.56	3.75	(32.56)
23	240	0.5	40.7	130	17	35.00	3.56	65.50	4.18	7.99	2.08	27	37.37	3.62	28.13
23	240	0.5	110.2	288	39	9.50	2.25	15.00	2.71	17.12	2.84	(8)	46.50	3.84	(31.50)
24	240	0.5	59.9	100	17	19.00	2.94	120.00	4.79	8.08	2.09	11	37.46	3.62	82.54
25	240	3.2	153.4	82	28	19.50	2.97	30.00	3.40	20.33	3.01	(1)	49.70	3.91	(19.70)
25	240	0.5	112.8	136	27	42.00	3.74	76.00	4.33	12.27	2.51	30	41.64	3.73	34.36
25	240	0.5	84.2	140	23	17.00	2.83	27.50	3.31	10.79	2.38	6	40.16	3.69	(12.66)
25	240	0.5	36.4	68	11	16.00	2.77	48.00	3.87	5.68	1.74	10	35.06	3.56	12.94
25	240	0.5	133.2	151	31	12.50	2.53	39.00	3.66	13.91	2.63	(1)	43.29	3.77	(4.29)
26	240	0.5	61.2	306	34	14.00	2.64	38.50	3.65	14.95	2.70	(1)	44.32	3.79	(5.82)
26	240	0.5	124.5	198	34	12.50	2.53	30.00	3.40	14.96	2.71	(2)	44.34	3.79	(14.34)
26	240	0.5	62.2	163	22	17.50	2.86	27.00	3.30	10.29	2.33	7	39.66	3.68	(12.66)
27	240	0.5	71.4	248	31	5.50	1.70	26.50	3.28	13.60	2.61	(8)	42.97	3.76	(16.47)
27	240	0.5	109.2	220	34	13.00	2.56	22.50	3.11	14.82	2.70	(2)	44.20	3.79	(21.70)
27	240	0.5	103.4	178	29	12.50	2.53	26.50	3.28	13.11	2.57	(1)	42.48	3.75	(15.98)
27	240	0.5	34.9	108	14	11.50	2.44	19.50	2.97	6.91	1.93	5	36.29	3.59	(16.79)
28	240	0.5	60.4	154	21	6.00	1.79	9.00	2.20	9.90	2.29	(4)	39.27	3.67	(30.27)
28	240	0.5	77.5	254	32	14.00	2.64	23.00	3.14	14.14	2.65	0	43.51	3.77	(20.51)
28	240	0.5	199.1	258	50	10.00	2.30	37.50	3.62	21.19	3.05	(11)	50.56	3.92	(13.06)
29	240	0.5	224.4	174	63	21.00	3.04	30.00	3.40	26.41	3.27	(5)	55.78	4.02	(25.78)
29	240	0.5	41.8	50	10	18.50	2.92	24.00	3.18	5.41	1.69	13	34.78	3.55	(10.78)
29	240	0.5	75.2	122	21	20.00	3.00	29.00	3.37	9.67	2.27	10	39.04	3.66	(10.04)
29	240	0.5	31.3	82	11	17.50	2.86	33.00	3.50	5.87	1.77	12	35.25	3.56	(2.25)
30	240	0.5	51.4	112	17	15.00	2.71	18.50	2.92	8.00	2.08	7	37.37	3.62	(18.87)
31	240	0.5	83.0	93	19	6.00	1.79	45.00	3.81	9.15	2.21	(3)	38.53	3.65	6.47
31	240	0.5	99.1	253	35	7.00	1.95	20.50	3.02	15.33	2.73	(8)	44.71	3.80	(24.21)
31	240	0.5	83.7	242	32	14.00	2.64	28.00	3.33	14.12	2.65	0	43.49	3.77	(15.49)
31	240	0.5	91.7		13	9.00	2.20	14.50	2.67	6.60	1.89	2	35.97	3.58	(21.47)
31	240	0.5	37.0	160	18	11.00	2.40	25.50	3.24	8.74	2.17	2	38.12	3.64	(12.62)
31	240	0.5	81.9	134	23	31.50				10.44		21	39.82		
32	240	0.5	52.0	186	23	9.50	2.25	19.00	2.94	10.48	2.35	(1)	39.85	3.69	(20.85)
32	240	0.5	63.1	145	21	15.00	2.71	25.50	3.24	9.75	2.28	5	39.12	3.67	(13.62)
32	240	8.9	74.7	166	24	12.00	2.48	19.00	2.94	34.59	3.54	(23)	63.96	4.16	(44.96)
32	240	0.5	73.0	83	17	18.00	2.89	21.00	3.04	8.25	2.11	10	37.63	3.63	(16.63)
32	240	0.5	224.4	374	63	10.00	2.30	18.00	2.89	26.41	3.27	(16)	55.78	4.02	(37.78)
33	240	0.5	65.8	190	25	16.00	2.77	21.00	3.04	11.38	2.43	5	40.76	3.71	(19.76)
33	240	0.5	49.9	97	15	13.50	2.60	23.50	3.16	7.41	2.00	6	36.79	3.61	(13.29)
33	240	0.5	16.1	159	15	11.50	2.44	22.00	3.09	7.53	2.02	4	36.91	3.61	(14.91)
33	240	0.5	56.3	224	26	8.50	2.14	16.00	2.77	11.97	2.48	(3)	41.35	3.72	(25.35)
33	240	0.5	48.0	92	14	10.50	2.35	15.50	2.74	7.15	1.97	3	36.52	3.60	(21.02)
33	240	0.5	93.2	243	33	9.50	2.25	20.50	3.02	14.66	2.69	(5)	44.04	3.79	(23.54)
33	240	0.5	49.8	217	25	7.50	2.01	30.00	3.40	11.35	2.43	(4)	40.72	3.71	(10.72)
33	240	2.9	121.8	194	33	14.00	2.64	35.00	3.56	21.41	3.06	(7)	50.78	3.93	(15.78)
33	240	0.5	45.0	84	13	7.50	2.01	21.00	3.04	6.71	1.90	1	36.08	3.59	(15.08)
34	240	0.5	277.9	275	62	28.50	3.35	47.50	3.86	26.22	3.27	2	55.59	4.02	(8.09)
34	240	0.5	42.7	165	20	6.00	1.79	12.00	2.48	9.24	2.22	(3)	38.62	3.65	(26.62)
34	240	1.4	125.6	82	25	10.50	2.35	27.00	3.30	13.74	2.62	(3)	43.12	3.76	(16.12)
34	240	0.5	50.4	171	21	6.00	1.79	12.00	2.48	9.89	2.29	(4)	39.26	3.67	(27.26)
34	240	0.5	154.0	303	47	42.50	3.75	68.50	4.23	20.11	3.00	22	49.48	3.90	19.02
34	240	0.5	246.1	289	59	18.00	2.89	29.00	3.37	24.87	3.21	(7)	54.24	3.99	(25.24)
34	240	0.5	58.5	260	30	5.50	1.70	12.00	2.48	13.26	2.58	(8)	42.63	3.75	(30.63)
35	240	0.5	0.0	321	26	13.00	2.56	22.00	3.09	11.95	2.48	1	41.32	3.72	(19.32)
35	240	0.5	144.3	189	36	13.00	2.56	25.00	3.22	15.80	2.76	(3)	45.17	3.81	(20.17)
35	240	0.5	52.9	174	22	5.50	1.70	17.00	2.83	10.12	2.31	(5)	39.50	3.68	(22.50)
35	240	0.5	94.7	228	32	9.00	2.20	18.00	2.89	14.27	2.66	(5)	43.64	3.78	(25.64)
35	240	2.0	30.5	69	10	5.50	1.70	32.50	3.48	9.60	2.26	(4)	38.97	3.66	(6.47)
35	240	1.4	42.8	43	10	8.50	2.14	19.50	2.97	7.77	2.05	1	37.14	3.61	(17.64)

TABLE 2
ACTUAL AND PREDICTED URINARY ARSENIC LEVELS

AGE IN MONTHS	ESTIMATED URINE PRODUCTION	ARSENIC IN WATER (ug/L)	ARSENIC IN DUST (mg/kg)	ARSENIC IN SOIL (mg/kg)	55% DUST 45% SOIL (mg/kg) ABSORBED	MEAN SPEC URINARY AS NORMALIZED (ug/L)	NATURAL LOG MEAN SPEC URINARY AS NORMALIZED	MEAN TOTAL URINARY AS NORMALIZED (ug/L)	NATURAL LOG MEAN TOTAL URINARY AS NORMALIZED	EPA PREDICTED SPECIATED AS CTE (ug/L)	NATURAL LOG EPA PREDICTED SPECIATED AS CTE (ug/L)	DIFFERENCE BETWEEN SPECIATED MEASURED AND PREDICTED	EPA PREDICTED TOTAL AS CTE (ug/L)	NATURAL LOG EPA PREDICTED TOTAL AS CTE (ug/L)	DIFFERENCE BETWEEN TOTAL MEASURED AND PREDICTED
35	240	0.5	105.3	106	24	20.00	3.00	39.00	3.66	10.86	2.39	9	40.24	3.69	(1.24)
35	240	0.5	49.3	138	18	22.00	3.09	29.50	3.38	8.73	2.17	13	38.10	3.64	(8.60)
35	240	0.5	102.1	259	36	12.50	2.53	25.50	3.24	15.69	2.75	(3)	45.07	3.81	(19.57)
35	240	0.5	43.5	163	20	23.00	3.14	25.00	3.22	9.24	2.22	14	38.61	3.65	(13.61)
36	240	0.5	125.1	268	40	5.50	1.70	13.50	2.60	17.30	2.85	(12)	46.68	3.84	(33.18)
36	240	0.5	125.5	179	33	10.00	2.30	16.00	2.77	14.40	2.67	(4)	43.78	3.78	(27.78)
36	240	0.5	135.6	187	35	8.00	2.08	14.50	2.67	15.23	2.72	(7)	44.60	3.80	(30.10)
36	355	0.5	14.9	128	13	9.00	2.20	20.00	3.00	4.36	1.47	5	24.22	3.19	(4.22)
36	355	0.5	40.7	123	16	13.50	2.60	20.00	3.00	5.24	1.66	8	25.10	3.22	(5.10)
37	355	0.5	69.9	162	23	9.00	2.20	27.50	3.31	7.23	1.98	2	27.09	3.30	0.41
37	355	0.5	36.4	68	11	8.00	2.08	43.00	3.76	3.84	1.35	4	23.70	3.17	19.30
37	355	0.5	104.7	228	34	16.00	2.77	33.50	3.51	10.03	2.31	6	29.89	3.40	3.61
37	355	0.5	54.3	258	29	11.50	2.44	18.00	2.89	8.76	2.17	3	28.62	3.35	(10.62)
37	355	0.5	84.4	149	24	9.00	2.20	10.00	2.30	7.49	2.01	2	27.35	3.31	(17.35)
37	355	0.5	101.8	265	36	6.00	1.79	28.00	3.33	10.73	2.37	(5)	30.59	3.42	(2.59)
37	355	0.5	119.7	193	33	14.00	2.64	37.00	3.61	9.83	2.29	4	29.69	3.39	7.31
38	355	1.6	149.8	96	29	18.00	2.89	43.50	3.77	10.89	2.39	7	30.75	3.43	12.75
38	355	4.7	43.3	62	11	20.00	3.00	36.50	3.60	11.93	2.48	8	31.78	3.46	4.72
38	355	2.9	49.2	72	13	25.00	3.22	39.00	3.66	8.97	2.19	16	28.83	3.36	10.17
38	355	0.5	103.4	118	24	15.00	2.71	21.00	3.04	7.54	2.02	7	27.40	3.31	(6.40)
38	355	0.5	49.4	73	13	8.50	2.14	135.00	4.91	4.45	1.49	4	24.31	3.19	110.69
38	355	0.5	53.6	151	20	11.00	2.40	20.50	3.02	6.36	1.85	5	26.22	3.27	(5.72)
38	355	0.5	190.2	239	47	24.50	3.20	51.00	3.93	13.55	2.61	11	33.41	3.51	17.59
38	355	0.5	237.9	248	54	11.50	2.44	20.00	3.00	15.57	2.75	(4)	35.43	3.57	(15.43)
38	355	0.5	39.7	81	12	21.00	3.04	31.00	3.43	4.26	1.45	17	24.12	3.18	6.88
39	355	0.5	85.3	313	38	11.00	2.40	22.50	3.11	11.17	2.41	0	31.03	3.43	(8.53)
39	355	0.5	70.5	161	23	11.50	2.44	25.00	3.22	7.23	1.98	4	27.09	3.30	(2.09)
39	355	0.5	69.8	205	27					8.18			28.03		
40	355	0.5	150.9	263	43	9.00	2.20	15.50	2.74	12.58	2.53	(4)	32.44	3.48	(16.94)
40	355	0.5	85.7	148	24	16.50	2.80	38.00	3.64	7.51	2.02	9	27.37	3.31	10.63
40	355	0.5	65.8	194	25	17.50	2.86	17.50	2.86	7.77	2.05	10	27.63	3.32	(10.13)
40	355	0.5	47.6	69	12	5.50	1.70	14.50	2.67	4.31	1.46	1	24.17	3.19	(9.67)
40	355	0.5	67.3	203	26	7.00	1.95	15.50	2.74	8.03	2.08	(1)	27.89	3.33	(12.39)
40	355	0.5	89.4	126	23	11.50	2.44	21.50	3.07	7.18	1.97	4	27.04	3.30	(5.54)
40	355	0.5	63.6	134	20	19.50	2.97	28.00	3.33	6.36	1.85	13	26.22	3.27	1.78
40	355	0.5	70.6	223	28	17.00	2.83	19.50	2.97	8.62	2.15	8	28.48	3.35	(8.98)
41	355	0.5	115.5	214	34	8.00	2.08	17.50	2.86	10.13	2.32	(2)	29.99	3.40	(12.49)
41	355	0.5	38.2	229	24	17.00	2.83	41.00	3.71	7.51	2.02	9	27.37	3.31	13.63
41	355	0.5	31.0	142	16	9.50	2.25	9.00	2.20	5.30	1.67	4	25.16	3.23	(16.16)
41	355	0.5	168.3	349	53	14.50	2.67	46.00	3.83	15.15	2.72	(1)	35.01	3.56	10.99
41	355	0.5	124.5	198	34	3.00	1.10	18.00	2.89	10.12	2.31	(7)	29.98	3.40	(11.98)
41	355	2.6	47.8	62	12	15.00	2.71	27.50	3.31	8.13	2.10	7	27.99	3.33	(0.49)
41	355	0.5	64.2	129	20	34.00	3.53	51.00	3.93	6.28	1.84	28	26.14	3.26	24.86
41	355	0.5	175.8	310	50	13.50	2.60	26.00	3.26	14.57	2.68	(1)	34.43	3.54	(8.43)
41	355	0.5	25.1	162	17					5.51			25.36		
41	355	0.5	151.6	341	50	11.50	2.44	34.00	3.53	14.33	2.66	(3)	34.19	3.53	(0.19)
41	355	0.5	98.5	158	27	6.00	1.79	18.50	2.92	8.24	2.11	(2)	28.09	3.34	(9.59)
41	355	0.5	129.8	240	38	18.50	2.92	29.50	3.38	11.25	2.42	7	31.11	3.44	(1.61)
42	355	0.5	110.8	187	31	9.00	2.20	31.50	3.45	9.36	2.24	0	29.22	3.37	2.28
42	355	3.9	17.8	80	9	8.00	2.08	17.00	2.83	9.84	2.29	(2)	29.70	3.39	(12.70)
42	355	0.5	81.0	151	24					7.42			27.28		
42	355	0.5	160.0	174	37	14.00	2.64	34.50	3.54	10.95	2.39	3	30.81	3.43	3.69
42	355	0.5	81.2	232	31	19.00	2.94	212.50	5.36	9.21	2.22	10	29.07	3.37	183.43
42	355	3.3	83.4	86	19	11.00	2.40	20.50	3.02	11.35	2.43	0	31.21	3.44	(10.71)
43	355	0.5	81.4	293	36	13.00	2.56	23.00	3.14	10.57	2.36	2	30.43	3.42	(7.43)
43	355	1.6	85.2	94	20	21.00	3.04	28.00	3.33	8.38	2.13	13	28.24	3.34	(0.24)
43	355	0.5	91.9	204	30	5.50	1.70	12.00	2.48	9.01	2.20	(4)	28.87	3.36	(16.87)
43	355	0.5	260.1	232	56	19.00	2.94	47.00	3.85	16.08	2.78	3	35.94	3.58	11.06
43	355	0.5	119.8	278	40	8.50	2.14	10.00	2.30	11.72	2.46	(3)	31.57	3.45	(21.57)
43	355	0.5	63.2	192	25	47.00	3.85	100.00	4.61	7.65	2.03	39	27.51	3.31	72.49
43	355	0.5	178.5	256	46	9.50	2.25	18.50	2.92	13.47	2.60	(4)	33.33	3.51	(14.83)
43	355	2.7	38.7	79	12	7.00	1.95	34.00	3.53	8.35	2.12	(1)	28.21	3.34	5.79
43	355	0.5	37.3	78	12	11.00	2.40	21.00	3.04	4.11	1.41	7	23.97	3.18	(2.97)

TABLE 2
ACTUAL AND PREDICTED URINARY ARSENIC LEVELS

AGE IN MONTHS	ESTIMATED URINE PRODUCTION	ARSENIC IN WATER (ug/L)	ARSENIC IN DUST (ng/kg)	ARSENIC IN SOIL (ng/kg)	55%DUST 45%SOIL (ng/kg) ABSORBED	MEAN SPEC URINARY AS NORMALIZED (ug/L)	NATURAL LOG MEAN SPEC URINARY AS NORMALIZED	MEAN TOTAL URINARY AS NORMALIZED (ug/L)	NATURAL LOG MEAN TOTAL URINARY AS NORMALIZED	EPA PREDICTED SPECIATED AS CTE (ug/L)	NATURAL LOG EPA PREDICTED SPECIATED AS CTE (ug/L)	DIFFERENCE BETWEEN SPECIATED MEASURED AND PREDICTED	EPA PREDICTED TOTAL AS CTE (ug/L)	NATURAL LOG EPA PREDICTED TOTAL AS CTE (ug/L)	DIFFERENCE BETWEEN TOTAL MEASURED AND PREDICTED
43	355	0.5	112.8	136	27	20.50	3.02	55.00	4.01	8.29	2.12	12	28.15	3.34	26.85
44	355	0.5	190.3	229	46	16.00	2.77	295.00	5.69	13.32	2.59	3	33.18	3.50	261.82
44	355	0.5	68.8	105	18	5.50	1.70	10.00	2.30	5.92	1.78	0	25.72	3.25	(15.78)
44	355	0.5	102.7	137	26	11.50	2.44	24.50	3.20	7.93	2.07	4	27.79	3.32	(3.29)
44	355	0.5	91.8	233	32	16.50	2.80	32.50	3.48	9.64	2.27	7	29.49	3.38	3.01
44	355	0.5	114.1	252	37	15.00	2.71	20.00	3.00	10.92	2.39	4	30.78	3.43	(10.78)
44	355	1.5	49.9	87	13	7.00	1.95	14.00	2.64	6.34	1.85	1	26.20	3.27	(12.20)
44	355	9.9	29.5	71	10	3.00	1.10	8.00	2.08	21.43	3.06	(18)	41.29	3.72	(33.29)
44	355	8.9	74.7	166	24	15.00	2.71	82.50	4.41	23.38	3.15	(8)	43.24	3.77	39.26
45	355	0.5	35.1	156	18	8.00	2.08	12.50	2.53	5.77	1.75	2	25.63	3.24	(13.13)
45	355	2.9	45.4	129	17	9.00	2.20	18.00	2.89	10.09	2.31	(1)	29.95	3.40	(11.95)
45	355	0.5	71.8	187	26	6.00	1.79	17.00	2.83	7.86	2.06	(2)	27.72	3.32	(10.72)
45	355	0.5	154.0	303	47	22.00	3.09	41.50	3.73	13.59	2.61	8	33.45	3.51	8.05
45	355	0.5	77.4	190	27	9.00	2.20	13.50	2.60	8.15	2.10	1	28.00	3.33	(14.50)
45	355	0.5	41.8	50	10	32.50	3.48	41.00	3.71	3.66	1.30	29	23.51	3.16	17.49
45	355	0.5	80.5	164	25	14.00	2.64	31.00	3.43	7.67	2.04	6	27.53	3.32	3.47
45	355	0.5	117.9	159	30	12.50	2.53	56.00	4.03	8.99	2.20	4	28.85	3.36	27.15
45	355	0.5	35.6	64	10	13.00	2.56	22.00	3.09	3.73	1.32	9	23.59	3.16	(1.59)
45	355	0.5	69.8	183	25	7.00	1.95	18.00	2.89	7.68	2.04	(1)	27.54	3.32	(9.54)
45	355	0.5	142.3	202	37	6.50	1.87	13.00	2.56	10.90	2.39	(4)	30.76	3.43	(17.76)
45	355	0.5	126.5	238	38	12.00	2.48	28.00	3.33	11.08	2.41	1	30.94	3.43	(2.94)
45	355	0.5	60.5	156	21	20.00	3.00	37.00	3.61	6.73	1.91	13	26.59	3.28	10.41
45	355	0.5	133.5	145	31	23.00	3.14	59.50	4.09	9.28	2.23	14	29.14	3.37	30.36
46	355	0.5	76.1	127	21	5.50	1.70	14.00	2.64	6.69	1.90	(1)	26.55	3.28	(12.55)
46	355	0.5	164.5	268	45	16.00	2.77	26.00	3.26	13.21	2.58	3	33.07	3.50	(7.07)
46	355	0.5	61.2	306	34	9.50	2.25	27.50	3.31	10.11	2.31	(1)	29.96	3.40	(2.46)
46	355	0.5	58.8	131	19	5.50	1.70	20.50	3.02	6.10	1.81	(1)	25.96	3.26	(5.46)
46	355	0.5	72.3	323	37	10.50	2.35	17.50	2.86	10.91	2.39	0	30.77	3.43	(13.27)
46	355	0.5	45.3	128	17	6.00	1.79	12.50	2.53	5.52	1.71	0	25.38	3.23	(12.88)
46	355	0.5	25.6	203	20	16.00	2.77	41.00	3.71	6.44	1.86	10	26.30	3.27	14.70
46	355	0.5	175.8	310	50	11.00	2.40	17.00	2.83	14.57	2.68	(4)	34.43	3.54	(17.43)
46	355	0.5	74.7	92	18	8.00	2.08	21.50	3.07	5.86	1.77	2	25.72	3.25	(4.22)
47	355	0.5	86.3	164	26	5.00	1.61	9.50	2.25	7.90	2.07	(3)	27.75	3.32	(18.25)
47	355	0.5	40.7	130	17	34.00	3.53	44.50	3.80	5.40	1.69	29	25.26	3.23	19.24
47	355	0.5	73.0	138	22	13.00	2.56	24.50	3.20	6.82	1.92	6	26.68	3.28	(2.18)
47	355	0.5	32.6	197	21	9.50	2.25	18.50	2.92	6.57	1.88	3	26.43	3.27	(7.93)
47	355	0.5	133.2	151	31	11.50	2.44	27.00	3.30	9.41	2.24	2	29.26	3.38	(2.26)
47	355	0.5	76.9	208	28	5.00	1.61	6.50	1.87	8.52	2.14	(4)	28.38	3.35	(21.88)
47	355	0.5	19.3	104	11	19.00	2.94	19.00	2.94	3.99	1.38	15	23.85	3.17	(4.85)
47	355	0.5	72.5	174	25	6.00	1.79	11.00	2.40	7.59	2.03	(2)	27.45	3.31	(16.45)
48	355	0.5	59.6	125	19	13.50	2.60	49.50	3.90	6.02	1.79	7	25.88	3.25	23.62
48	355	0.5	23.9	107	12	8.50	2.14	10.00	2.30	4.25	1.45	4	24.11	3.18	(14.11)
48	355	0.5	145.4	140	32	9.00	2.20	19.50	2.97	9.64	2.27	(1)	29.50	3.38	(10.00)
48	355	0.5	64.7	119	19	8.50	2.14	20.50	3.02	6.07	1.80	2	25.93	3.26	(5.43)
48	355	0.5	135.6	187	35	7.50	2.01	22.00	3.09	10.29	2.33	(3)	30.15	3.41	(8.15)
48	355	0.5	25.2	159	17	14.00	2.64	31.00	3.43	5.44	1.69	9	25.30	3.23	5.70
48	355	0.5	146.4	311	46	14.00	2.64	28.00	3.33	13.46	2.60	1	33.32	3.51	(5.32)
48	355	0.5	49.2	140	18	11.00	2.40	26.50	3.28	5.93	1.78	5	25.79	3.25	0.71
49	355	0.5	82.2	209	29	18.00	2.89	16.50	2.80	8.74	2.17	9	28.60	3.35	(12.10)
49	355	2.2	48.3	63	12	5.50	1.70	12.50	2.53	7.41	2.00	(2)	27.27	3.31	(14.77)
49	355	0.5	83.3	360	41	5.00	1.61	13.00	2.56	12.14	2.50	(7)	32.00	3.47	(19.00)
49	355	0.5	73.0	202	27	6.00	1.79	55.50	4.02	8.24	2.11	(2)	28.10	3.34	27.40
49	355	0.5	157.1	266	44	9.50	2.25	13.00	2.56	12.89	2.56	(3)	32.75	3.49	(19.75)
49	355	3.3	25.2	66	9	5.50	1.70	26.00	3.26	8.67	2.16	(3)	28.53	3.35	(2.53)
50	355	0.5	122.2	142	29	8.00	2.08	23.00	3.14	8.80	2.17	(1)	28.65	3.36	(5.65)
50	355	0.5	39.9	103	14	6.00	1.79	27.50	3.31	4.77	1.56	1	24.63	3.20	2.87
50	355	0.5	42.4	138	17	7.50	2.01	6.00	1.79	5.63	1.73	2	25.49	3.24	(19.49)
50	355	0.5	191.3	294	51	9.50	2.25	14.50	2.67	14.81	2.70	(5)	34.67	3.55	(20.17)
50	355	0.5	63.7	106	18	8.00	2.08	17.00	2.83	5.75	1.75	2	25.61	3.24	(8.61)
50	355	2.2	95.7	84	20	13.00	2.56	25.00	3.22	9.69	2.27	3	29.54	3.39	(4.54)
51	355	0.5	11.9	215	19	13.50	2.60	26.50	3.28	6.18	1.82	7	26.04	3.26	0.46
51	355	0.5	202.5	276	51	11.50	2.44	17.50	2.86	14.86	2.70	(3)	34.71	3.55	(17.21)
51	355	0.5	91.5	226	32	7.50	2.01	22.00	3.09	9.48	2.25	(2)	29.34	3.38	(7.34)

TABLE 2
ACTUAL AND PREDICTED URINARY ARSENIC LEVELS

AGE IN MONTHS	ESTIMATED URINE PRODUCTION	ARSENIC IN WATER (ug/L)	ARSENIC IN DUST (ug/kg)	ARSENIC IN SOIL (ug/kg)	55%DUST 45%SOIL (ug/kg)	MEAN SPEC URINARY AS NORMALIZED (ug/L)	NATURAL LOG MEAN SPEC URINARY AS NORMALIZED	MEAN TOTAL URINARY AS NORMALIZED (ug/L)	NATURAL LOG MEAN TOTAL URINARY AS NORMALIZED	EPA PREDICTED SPECIATED AS CTE (ug/L)	NATURAL LOG EPA PREDICTED SPECIATED AS CTE (ug/L)	DIFFERENCE BETWEEN SPECIATED MEASURED AND PREDICTED	EPA PREDICTED TOTAL AS CTE (ug/L)	NATURAL LOG EPA PREDICTED TOTAL AS CTE (ug/L)	DIFFERENCE BETWEEN TOTAL MEASURED AND PREDICTED
51	355	0.5	82.5	214	29	13.50	2.60	37.50	3.62	8.86	2.18	5	28.72	3.36	8.78
52	355	1.2	127.8	101	26	9.50	2.25	17.50	2.86	9.42	2.24	0	29.28	3.38	(11.78)
52	355	0.5	75.5	314	37	8.00	2.08	26.00	3.26	10.82	2.38	(3)	30.68	3.42	(4.68)
52	355	0.5	73.5	138	22	6.50	1.87	23.00	3.14	6.84	1.92	0	26.70	3.28	(3.70)
52	355	0.5	109.2	220	34	12.00	2.48	15.00	2.71	10.02	2.30	2	29.88	3.40	(14.88)
52	355	0.5	67.8	119	19	8.50	2.14	94.50	4.55	6.19	1.82	2	26.04	3.26	68.46
52	355	1.1	26.1	87	11	15.00	2.71	30.50	3.42	5.01	1.61	10	24.87	3.21	5.63
52	355	1.5	51.5	62	12	6.50	1.87	20.00	3.00	6.19	1.82	0	26.05	3.26	(6.05)
53	355	0.5	33.6	81	11	10.00	2.30	20.00	3.00	4.02	1.39	6	23.88	3.17	(3.88)
53	355	0.5	33.3	257	26	6.50	1.87	10.50	2.35	7.94	2.07	(1)	27.80	3.32	(17.30)
53	355	0.5	136.6	153	32	17.00	2.83	32.00	3.47	9.59	2.26	7	29.45	3.38	2.55
53	355	0.5	29.7	208	21	8.50	2.14	18.00	2.89	6.72	1.90	2	26.58	3.28	(8.58)
54	355	0.5	147.0	254	42	5.00	1.61	12.50	2.53	12.23	2.50	(7)	32.09	3.47	(19.59)
54	355	0.5	22.6	128	14	9.00	2.20	6.50	1.87	4.67	1.54	4	24.53	3.20	(18.03)
54	355	0.5	237.9	248	54	11.50	2.44	18.00	2.89	15.57	2.75	(4)	35.43	3.57	(17.43)
54	355	0.5	23.6	127	14	9.00	2.20	22.00	3.09	4.67	1.54	4	24.53	3.20	(2.53)
54	355	0.5	74.0	269	33	9.50	2.25	16.50	2.80	9.76	2.28	0	29.62	3.39	(13.12)
54	355	0.5	62.3	218	27	6.00	1.79	16.50	2.80	8.17	2.10	(2)	28.03	3.33	(11.53)
54	355	2.3	84.1	88	19	4.00	1.39	7.00	1.95	9.52	2.25	(6)	29.38	3.38	(22.38)
55	355	0.5	70.9	184	25	13.00	2.56	12.50	2.53	7.76	2.05	5	27.61	3.32	(15.11)
55	355	0.5	53.6	151	20	8.50	2.14	28.50	3.35	6.36	1.85	2	26.22	3.27	2.28
55	355	0.5	59.9	100	17	9.00	2.20	80.50	4.39	5.47	1.70	4	25.32	3.23	55.18
55	355	3.5	56.1	127	18	7.00	1.95	16.00	2.77	11.58	2.45	(5)	31.44	3.45	(15.44)
55	355	4.0	23.7	79	10	8.00	2.08	16.00	2.77	10.22	2.32	(2)	30.08	3.40	(14.08)
55	355	0.5	124.5	198	34	3.00	1.10	26.00	3.26	10.12	2.31	(7)	29.98	3.40	(3.98)
56	355	0.5	38.2	57	10	5.00	1.61	18.50	2.92	3.68	1.30	1	23.54	3.16	(5.04)
56	355	0.5	174.1	197	41	9.00	2.20	15.50	2.74	12.00	2.48	(3)	31.86	3.46	(16.36)
56	355	0.5	79.6	212	29	7.50	2.01	19.00	2.94	8.71	2.16	(1)	28.57	3.35	(9.57)
56	355	0.5	50.0	59	12	5.50	1.70	14.50	2.67	4.18	1.43	1	24.04	3.18	(9.54)
56	355	0.5	101.3	272	37	70.50	4.26	78.50	4.36	10.87	2.39	60	30.73	3.43	47.77
56	355	0.5	45.5	202	23	10.00	2.30	18.00	2.89	7.19	1.97	3	27.05	3.30	(9.05)
56	355	0.5	32.9	106	13	4.50	1.50	20.00	3.00	4.56	1.52	0	24.42	3.20	(4.42)
56	355	0.5	101.0	179	29	10.50	2.35	17.00	2.83	8.80	2.17	2	28.66	3.36	(11.66)
56	355	0.5	73.5	64	16	7.00	1.95	14.50	2.67	5.19	1.65	2	25.05	3.22	(10.55)
56	355	0.5	99.1	253	35	6.00	1.79	16.50	2.80	10.37	2.34	(4)	30.23	3.41	(13.73)
57	355	0.5	62.7	102	17	7.50	2.01	15.00	2.71	5.61	1.73	2	25.47	3.24	(10.47)
57	355	0.5	178.5	256	46	7.50	2.01	24.00	3.18	13.47	2.60	(6)	33.33	3.51	(9.33)
57	355	0.5	19.7	110	12	10.00	2.30	72.50	4.28	4.14	1.42	6	24.00	3.18	48.50
57	355	0.5	68.8	174	24	13.00	2.56	15.50	2.74	7.45	2.01	6	27.31	3.31	(11.81)
57	355	0.5	124.8	261	39	11.50	2.44	18.00	2.89	11.53	2.44	0	31.39	3.45	(13.39)
57	355	0.5	0.0	120	10	7.00	1.95	10.50	2.35	3.62	1.29	3	23.48	3.16	(12.98)
57	355	0.5	49.8	217	25	7.00	1.95	21.50	3.07	7.67	2.04	(1)	27.53	3.32	(6.03)
57	355	0.5	70.7	121	20	8.00	2.08	34.50	3.54	6.34	1.85	2	26.20	3.27	8.30
58	355	0.5	119.3	136	28	8.50	2.14	46.00	3.83	8.55	2.15	0	28.41	3.35	17.59
58	355	0.5	156.3	215	40	12.00	2.48	18.50	2.92	11.71	2.46	0	31.57	3.45	(13.07)
58	355	0.5	70.5	161	23	8.50	2.14	21.50	3.07	7.23	1.98	1	27.09	3.30	(5.59)
58	355	0.5	45.9	143	18	10.50	2.35	8.00	2.08	5.89	1.77	5	25.75	3.25	(17.75)
58	355	1.5	38.4	56	10	8.00	2.08	22.00	3.09	5.56	1.72	2	25.42	3.21	(3.42)
58	355	0.5	107.4	162	29	10.00	2.30	24.50	3.20	8.67	2.16	1	28.53	3.35	(4.03)
58	355	0.5	224.4	374	63	9.00	2.20	14.00	2.64	17.85	2.88	(9)	37.71	3.63	(23.71)
58	355	0.5	59.6	125	19	12.00	2.48	31.00	3.43	6.02	1.79	6	25.88	3.25	5.12
58	355	0.5	103.4	118	24	13.50	2.60	12.50	2.53	7.54	2.02	6	27.40	3.31	(14.90)
58	355	0.5	110.1	180	30	9.50	2.25	24.50	3.20	9.18	2.22	0	29.04	3.37	(4.54)
58	355	0.5	42.2	117	16	8.50	2.14	71.50	4.27	5.17	1.64	3	25.03	3.22	46.47
58	355	0.5	36.4	68	11	18.50	2.92	22.00	3.09	3.84	1.35	15	23.70	3.17	(1.70)
59	355	2.9	49.2	72	13	33.00	3.50	48.50	3.88	8.97	2.19	24	28.83	3.36	19.67
59	355	0.5	118.6	305	42	7.50	2.01	19.00	2.94	12.26	2.51	(5)	32.12	3.47	(13.12)
59	355	0.5	34.8	118	15	9.00	2.20	8.50	2.14	4.91	1.59	4	24.77	3.21	(16.27)
59	355	0.5	55.5	186	23					7.21			27.07		
59	355	0.5	85.6	83	19	5.00	1.61	11.00	2.40	6.07	1.80	(1)	25.92	3.26	(14.92)
59	355	0.5	90.3	221	31	4.50	1.50	13.50	2.60	9.32	2.23	(5)	29.18	3.37	(15.68)
59	355	0.5	94.5	212	31	10.00	2.30	68.50	4.23	9.27	2.23	1	29.13	3.37	39.37
59	355	0.5	62.2	163	22	5.50	1.70	8.50	2.14	6.96	1.94	(1)	26.81	3.29	(18.31)

TABLE 2
ACTUAL AND PREDICTED URINARY ARSENIC LEVELS

AGE IN MONTHS	ESTIMATED URINE PRODUCTION	ARSENIC IN WATER (ug/L)	ARSENIC IN DUST (mg/kg)	ARSENIC IN SOIL (mg/kg)	55%DUST 45%SOIL (mg/kg) ABSORBED	MEAN SPEC URINARY AS NORMALIZED (ug/L)	NATURAL LOG MEAN SPEC URINARY AS NORMALIZED	MEAN TOTAL URINARY AS NORMALIZED (ug/L)	NATURAL LOG MEAN TOTAL URINARY AS NORMALIZED	EPA PREDICTED SPECIATED AS CTE (ug/L)	NATURAL LOG EPA PREDICTED SPECIATED AS CTE (ug/L)	DIFFERENCE BETWEEN SPECIATED MEASURED AND PREDICTED	EPA PREDICTED TOTAL AS CTE (ug/L)	NATURAL LOG EPA PREDICTED TOTAL AS CTE (ug/L)	DIFFERENCE BETWEEN TOTAL MEASURED AND PREDICTED
60	355	0.5	104.6	249	35	9.00	2.20	14.00	2.64	10.50	2.35	(2)	30.36	3.41	(16.36)
60	355	0.5	52.0	186	23	8.00	2.08	16.00	2.77	7.08	1.96	1	26.94	3.29	(10.94)
60	355	2.0	30.5	69	10	5.00	1.61	32.50	3.48	6.49	1.87	(1)	26.35	3.27	6.15
60	355	0.5	81.2	232	31	16.00	2.77	100.50	4.61	9.21	2.22	7	29.07	3.37	71.43
60	432	0.5	60.4	154	21	7.50	2.01	23.50	3.16	5.50	1.70	2	21.82	3.08	1.68
60	432	0.5	34.0	69	10	3.50	1.25	108.50	4.69	3.11	1.13	0	19.42	2.97	89.08
61	432	0.5	97.6	112	23	6.50	1.87	5.50	1.70	5.89	1.77	1	22.21	3.10	(16.71)
61	432	0.5	97.6	112	23	8.50	2.14	6.00	1.79	5.89	1.77	3	22.21	3.10	(16.21)
61	432	2.3	60.8	72	15	8.50	2.14	9.00	2.20	6.80	1.92	2	23.12	3.14	(14.12)
61	432	0.5	56.4	250	29	9.50	2.25	19.50	2.97	7.12	1.96	2	23.44	3.15	(3.94)
61	432	0.5	105.6	162	28	10.00	2.30	14.00	2.64	7.06	1.95	3	23.37	3.15	(9.37)
61	432	0.5	70.1	122	20	8.50	2.14	93.50	4.54	5.21	1.65	3	21.53	3.07	71.97
61	432	0.5	43.5	163	20	11.50	2.44	14.00	2.64	5.13	1.64	6	21.45	3.07	(7.45)
61	432	0.5	210.7	171	44	6.50	1.87	20.50	3.02	10.54	2.36	(4)	26.86	3.29	(6.36)
62	432	0.5	393.6	272	78	15.00	2.71	36.50	3.60	18.15	2.90	(3)	34.47	3.54	2.03
62	432	0.5	81.9	134	23	5.50	1.70	14.50	2.67	5.80	1.76	0	22.12	3.10	(7.62)
62	432	0.5	73.0	138	22	11.50	2.44	20.50	3.02	5.60	1.72	6	21.92	3.09	(1.42)
62	432	0.5	170.3	321	51	10.00	2.30	21.00	3.04	12.01	2.49	(2)	28.33	3.34	(7.33)
62	432	0.5	67.0	341	38	13.00	2.56	24.50	3.20	9.11	2.21	4	25.43	3.24	(6.93)
62	432	0.5	50.9	176	22	12.50	2.53	31.50	3.45	5.59	1.72	7	21.91	3.09	9.59
62	432	0.5	59.9	141	20	5.00	1.61	9.00	2.20	5.25	1.66	0	21.56	3.07	(12.56)
62	432	0.5	38.2	229	24	8.50	2.14	26.00	3.26	6.17	1.82	2	22.49	3.11	3.51
62	432	0.5	60.0	98	17	7.50	2.01	15.50	2.74	4.47	1.50	3	20.78	3.03	(5.28)
62	432	0.5	92.2	237	33	9.50	2.25	19.50	2.97	8.01	2.08	1	24.33	3.19	(4.83)
62	432	0.5	49.5	187	22	9.00	2.20	9.00	2.20	5.75	1.75	3	22.07	3.09	(13.07)
63	432	0.5	190.2	239	47	7.00	1.95	13.50	2.60	11.13	2.41	(4)	27.45	3.31	(13.95)
63	432	0.5	63.7	199	25	6.00	1.79	14.50	2.67	6.43	1.86	0	22.74	3.12	(8.24)
63	432	0.5	33.6	81	11	5.50	1.70	15.50	2.74	3.31	1.20	2	19.63	2.98	(4.13)
63	432	0.5	132.0	305	44	14.50	2.67	33.50	3.51	10.52	2.35	4	26.84	3.29	6.66
64	432	0.5	42.7	165	20	9.00	2.20	14.00	2.64	5.13	1.64	4	21.45	3.07	(7.45)
64	432	0.5	25.1	162	17	16.00	2.77	14.50	2.67	4.52	1.51	11	20.84	3.04	(6.34)
64	432	3.3	83.4	86	19	7.00	1.95	26.50	3.28	9.33	2.23	(2)	25.65	3.24	9.85
64	432	0.5	64.4	100	17	5.00	1.61	14.50	2.67	4.63	1.53	0	20.95	3.04	(6.45)
64	432	0.5	109.6	226	33	5.00	1.61	16.50	2.80	8.08	2.09	(3)	24.40	3.19	(7.90)
64	432	0.5	114.5	134	27	6.00	1.79	12.50	2.53	6.84	1.92	(1)	23.16	3.14	(10.66)
64	432	0.5	114.5	134	27	7.00	1.95	14.00	2.64	6.84	1.92	0	23.16	3.14	(9.16)
65	432	0.5	95.6	189	29	8.50	2.14	11.50	2.44	7.25	1.98	1	23.57	3.16	(12.07)
65	432	0.5	48.0	124	17	6.00	1.79	16.00	2.77	4.55	1.52	1	20.87	3.04	(4.87)
65	432	0.5	62.7	203	26	12.00	2.48	29.00	3.37	6.46	1.87	6	22.78	3.13	6.22
65	432	0.5	62.7	203	26	11.50	2.44	30.00	3.40	6.46	1.87	5	22.78	3.13	7.22
65	432	0.5	41.1	96	14	8.50	2.14	15.50	2.74	3.82	1.34	5	20.14	3.00	(4.64)
65	432	0.5	136.6	153	32	14.50	2.67	25.00	3.22	7.88	2.06	7	24.20	3.19	0.80
65	432	0.5	73.7	191	26	6.00	1.79	72.50	4.28	6.59	1.89	(1)	22.91	3.13	49.59
65	432	0.5	25.2	91	11	2.50	0.92	9.50	2.25	3.23	1.17	(1)	19.55	2.97	(10.05)
65	432	0.5	49.4	73	13	8.00	2.08	170.50	5.14	3.66	1.30	4	19.98	2.99	150.52
65	432	0.5	103.6	147	27	7.00	1.95	18.00	2.89	6.73	1.91	0	23.05	3.14	(5.05)
65	432	0.5	63.2	146	21	6.00	1.79	14.50	2.67	5.44	1.69	1	21.76	3.08	(7.26)
66	432	0.5	67.3	203	26	10.00	2.30	13.00	2.56	6.60	1.89	3	22.92	3.13	(9.92)
66	432	0.5	72.5	174	25	5.00	1.61	10.00	2.30	6.24	1.83	(1)	22.56	3.12	(12.56)
66	432	0.5	114.4	149	28	8.00	2.08	10.00	2.30	7.10	1.96	1	23.42	3.15	(13.42)
66	432	0.5	164.5	268	45	13.50	2.60	16.50	2.80	10.86	2.38	3	27.18	3.30	(10.68)
66	432	0.5	69.8	183	25	5.00	1.61	13.00	2.56	6.31	1.84	(1)	22.63	3.12	(9.63)
66	432	2.7	74.4	164	24	6.00	1.79	9.00	2.20	9.54	2.26	(4)	25.86	3.25	(16.86)
66	432	0.5	102.1	259	36	9.50	2.25	30.50	3.42	8.72	2.17	1	25.04	3.22	5.46
66	432	0.5	52.2	109	16	7.50	2.01	12.00	2.48	4.41	1.48	3	20.73	3.03	(8.73)
67	432	0.5	59.0	214	26	9.50	2.25	14.00	2.64	6.54	1.88	3	22.86	3.13	(8.86)
67	432	0.5	51.6	169	21	7.00	1.95	13.00	2.56	5.49	1.70	2	21.80	3.08	(8.80)
67	432	1.6	106.0	132	26	6.50	1.87	22.00	3.09	8.24	2.11	(2)	24.56	3.29	(2.56)
67	432	0.5	124.5	198	34	3.00	1.10	15.00	2.71	8.31	2.12	(5)	24.63	3.20	(9.63)
67	432	0.5	129.0	163	32	8.50	2.14	14.50	2.67	7.82	2.06	1	24.14	3.18	(9.64)
67	432	9.9	29.5	71	10	4.00	1.39	8.50	2.14	37.40	3.62	(33)	33.93	3.52	(25.43)
67	432	0.5	119.7	193	33	22.00	3.09	26.50	3.28	8.08	2.09	14	24.40	3.19	2.10
67	432	0.5	56.9	77	14	6.50	1.87	9.50	2.25	3.97	1.38	3	20.29	3.01	(10.79)

TABLE 2
ACTUAL AND PREDICTED URINARY ARSENIC LEVELS

AGE IN MONTHS	ESTIMATED URINE PRODUCTION	ARSENIC IN WATER (ug/L)	ARSENIC IN DUST (ug/kg)	ARSENIC IN SOIL (ug/kg)	55% DUST 45% SOIL (ug/kg)	MEAN SPEC URINARY AS NORMALIZED (ug/L)	NATURAL LOG MEAN SPEC URINARY AS NORMALIZED	MEAN TOTAL URINARY AS NORMALIZED (ug/L)	NATURAL LOG MEAN TOTAL URINARY AS NORMALIZED	EPA PREDICTED SPECIATED AS CTE (ug/L)	NATURAL LOG EPA PREDICTED SPECIATED AS CTE (ug/L)	DIFFERENCE BETWEEN SPECIATED MEASURED AND PREDICTED	EPA PREDICTED TOTAL AS CTE (ug/L)	NATURAL LOG EPA PREDICTED TOTAL AS CTE (ug/L)	DIFFERENCE BETWEEN TOTAL MEASURED AND PREDICTED
67	432	0.5	48.3	122	17	5.00	1.61	13.50	2.60	4.53	1.51	0	20.85	3.04	(7.35)
68	432	0.5	62.7	102	17	7.50	2.01	15.50	2.74	4.61	1.53	3	20.93	3.04	(5.43)
68	432	0.5	29.0	212	22	9.00	2.20	19.00	2.94	5.56	1.72	3	21.88	3.09	(2.88)
68	432	1.1	85.5	150	24					7.13			23.45		
68	432	0.5	159.6	213	40	6.50	1.87	14.50	2.67	9.69	2.27	(3)	26.01	3.26	(11.51)
68	432	0.5	126.2	137	29	3.50	1.25	9.50	2.25	7.25	1.98	(4)	23.57	3.16	(14.07)
68	432	0.5	86.3	164	26	4.50	1.50	10.50	2.35	6.49	1.87	(2)	22.81	3.13	(12.31)
68	432	0.5	150.7	240	41	17.50	2.86	36.00	3.58	9.90	2.29	8	26.22	3.27	9.78
68	432	0.5	99.3	166	28	6.50	1.87	14.00	2.64	6.95	1.94	0	23.27	3.15	(9.27)
69	432	0.5	142.3	202	37	4.00	1.39	8.50	2.14	8.95	2.19	(5)	25.27	3.23	(16.77)
69	432	1.4	23.4	38	6	4.00	1.39	17.50	2.86	3.61	1.28	0	19.93	2.99	(2.43)
69	432	0.5	47.4	87	14	6.50	1.87	6.00	1.79	3.85	1.35	3	20.17	3.00	(14.17)
69	432	0.5	46.1	94	14	6.00	1.79	21.00	3.04	3.95	1.37	2	20.27	3.01	0.73
69	432	2.9	47.6	108	16	7.50	2.01	14.00	2.64	7.97	2.08	0	24.29	3.19	(10.29)
70	432	1.1	104.7	70	21	8.00	2.08	13.00	2.56	6.29	1.84	2	22.61	3.12	(9.61)
70	432	0.5	48.7	115	16	4.50	1.50	14.50	2.67	4.42	1.49	0	20.74	3.03	(6.24)
70	432	0.5	206.3	140	41	6.00	1.79	9.50	2.25	9.83	2.29	(4)	26.15	3.26	(16.65)
70	432	0.5	39.1	152	18	9.50	2.25	9.00	2.20	4.79	1.57	5	21.11	3.05	(12.11)
70	432	0.5	77.3	127	21	7.00	1.95	11.50	2.44	5.54	1.71	1	21.86	3.08	(10.36)
70	432	0.5	180.5	227	44	12.50	2.53	23.00	3.14	10.61	2.36	2	26.93	3.29	(3.93)
70	432	0.5	63.6	134	20	11.50	2.44	18.50	2.92	5.23	1.65	6	21.55	3.07	(3.05)
70	432	0.5	60.5	156	21	9.50	2.25	22.00	3.09	5.53	1.71	4	21.85	3.08	0.15
71	432	0.5	83.3	360	41	6.50	1.87	15.50	2.74	9.98	2.30	(3)	26.30	3.27	(10.80)
71	432	1.8	58.0	87	15	2.50	0.92	6.50	1.87	6.21	1.83	(4)	22.53	3.11	(16.03)
71	432	0.5	108.7	202	32	8.00	2.08	24.00	3.18	7.89	2.07	0	24.21	3.19	(0.21)
71	432	0.5	76.1	127	21	5.50	1.70	15.00	2.71	5.50	1.70	0	21.82	3.08	(6.82)
71	432	0.5	95.6	217	31	7.50	2.01	12.50	2.53	7.75	2.05	0	24.07	3.18	(11.57)
71	432	2.6	140.1	90	27	5.50	1.70	10.00	2.30	10.10	2.31	(5)	26.42	3.27	(16.42)
71	432	0.5	97.1	290	38	7.00	1.95	12.00	2.48	9.13	2.21	(2)	25.45	3.24	(13.45)
71	432	0.5	98.7	213	32	4.50	1.50	12.50	2.53	7.78	2.05	(3)	24.09	3.18	(11.59)
71	432	0.5	65.8	161	23					5.78			22.10		
71	432	0.5	95.2	270	36	7.00	1.95	16.00	2.77	8.70	2.16	(2)	25.02	3.22	(9.02)
72	432	0.5	83.7	242	32	8.50	2.14	11.50	2.44	7.84	2.06	1	24.16	3.18	(12.66)
72	432	0.5	101.3	272	37	22.00	3.09	39.00	3.66	8.93	2.19	13	25.25	3.23	13.75
72	432	0.5	132.5	128	29	5.50	1.70	8.00	2.08	7.28	1.99	(2)	23.60	3.16	(15.60)
72	432	0.5	110.1	180	30	5.50	1.70	12.00	2.48	7.54	2.02	(2)	23.86	3.17	(11.86)
72	432	2.6	42.0	60	11	4.00	1.39	9.00	2.20	6.46	1.87	(2)	22.78	3.13	(13.78)
72	432	0.5	126.5	301	43	13.00	2.56	29.00	3.37	10.26	2.33	3	26.58	3.28	2.42
72	432	0.5	182.3	238	45	9.00	2.20	12.50	2.53	10.87	2.39	(2)	27.19	3.30	(14.69)
73	432	0.5	284.4	300	65	7.00	1.95	7.50	2.01	15.21	2.72	(8)	31.53	3.45	(24.03)
73	432	0.5	107.6	120	25	4.00	1.39	10.00	2.30	6.37	1.85	(2)	22.69	3.12	(12.69)
73	432	0.5	60.6	89	16	5.50	1.70	12.50	2.53	4.31	1.46	1	20.63	3.03	(8.13)
73	432	1.4	71.7	134	21	4.50	1.50	8.00	2.08	6.87	1.93	(2)	23.19	3.14	(15.19)
73	432	0.5	46.2	88	14	4.50	1.50	9.50	2.25	3.84	1.34	1	20.16	3.00	(10.66)
73	432	0.5	129.8	240	38	11.50	2.44	36.00	3.58	9.25	2.22	2	25.57	3.24	10.43
74	432	0.5	38.2	57	10	6.00	1.79	22.00	3.09	3.03	1.11	3	19.35	2.96	2.65
74	432	0.5	35.5	213	23	6.50	1.87	12.50	2.53	5.79	1.76	1	22.11	3.10	(9.61)
74	432	0.5	55.2	120	18	11.00	2.40	16.50	2.80	4.71	1.55	6	21.03	3.05	(4.53)
74	432	0.5	138.7	309	45	5.00	1.61	14.00	2.64	10.80	2.38	(6)	27.12	3.30	(13.12)
74	432	0.5	205.4	139	41	8.00	2.08	28.50	3.35	9.79	2.28	(2)	26.11	3.26	2.39
74	432	0.5	153.8	280	45	5.50	1.70	10.00	2.30	10.74	2.37	(5)	27.06	3.30	(17.06)
75	432	0.5	43.2	84	13	11.00	2.40	15.50	2.74	3.68	1.30	7	20.00	3.00	(4.50)
75	432	0.5	333.4	409	81	8.00	2.08	13.50	2.60	18.76	2.93	(11)	35.08	3.56	(21.58)
75	432	0.5	32.6	197	21	5.50	1.70	14.50	2.67	5.40	1.69	0	21.72	3.08	(7.22)
76	432	0.5	122.1	131	28	6.00	1.79	50.00	3.91	7.01	1.95	(1)	23.33	3.15	26.67
76	432	0.5	75.1	157	24	6.50	1.87	12.50	2.53	6.01	1.79	0	22.33	3.11	(9.83)
76	432	0.5	74.2	44	14	2.50	0.92	14.00	2.64	3.92	1.37	(1)	20.24	3.01	(6.24)
76	432	0.5	51.6	127	18	3.00	1.10	7.00	1.95	4.72	1.55	(2)	21.04	3.05	(14.04)

TABLE 3

Urinary Arsenic Levels	Arithmetic Mean and Standard Deviation	Geometric Mean and Standard Deviation
Measured Speciated Urinary Arsenic	10.9 ± 7.1	9.4 ± 1.7
Predicted Speciated Urinary Arsenic	9.0 ± 4.4	8.1 ± 1.5
Measured Total Urinary Arsenic	26.1 ± 25.9	20.8 ± 1.8
Predicted Total Urinary Arsenic	29.2 ± 7.6	28.4 ± 1.3

TABLE 4
REASONABLE MAXIMUM EXPOSURE CALCULATIONS FOR RISKS IN COMMUNITY SOILS OU

		Dust Mean	Soil Mean	Risk	Bioavailability assumption
Area A	count	59	59	4.89E-05	assumes 25.8% bioavailability for dust; 18.3% bioavailability for soil
	mean	56.11	99.91		
	standard deviation	30.95	41.10		
	95% u c l of mean	62.85	108.86		
Area B	count	70	70	6.40E-05	assumes 25.8% bioavailability for dust; 18.3% bioavailability for soil
	mean	65.97	145.79		
	standard deviation	40.82	53.81		
	95% u c l of mean	74.12	156.53		
Area C	count	36	37	7.72E-05	assumes 25.8% bioavailability for dust; 18.3% bioavailability for soil
	mean	59.56	194.03		
	standard deviation	66.47	51.31		
	95% u c l of mean	78.29	208.28		
Area D	count	23	23	1.15E-04	assumes 25.8% bioavailability for dust; 18.3% bioavailability for soil
	mean	114.20	256.34		
	standard deviation	43.79	84.04		
	95% u c l of mean	129.88	286.43		
Area E	count	68	68	8.87E-05	assumes 25.8% bioavailability for dust; 18.3% bioavailability for soil
	mean	87.55	219.80		
	standard deviation	37.69	51.97		
	95% u c l of mean	95.18	230.32		
Area F	count	135	135	1.10E-04	assumes 25.8% bioavailability for dust; 18.3% bioavailability for soil
	mean	118.99	255.75		
	standard deviation	63.15	76.97		
	95% u c l of mean	127.93	266.64		
Area G	count	28	28	7.41E-05	assumes 25.8% bioavailability for dust; 18.3% bioavailability for soil
	mean	79.44	150.32		
	standard deviation	40.41	61.62		
	95% u c l of mean	92.44	170.16		
Area H	count	17	17	6.06E-05	assumes 25.8% bioavailability for dust; 18.3% bioavailability for soil
	mean	70.19	103.46		
	standard deviation	33.41	47.94		
	95% u c l of mean	84.33	123.77		
Area I	count	12	12	8.05E-05	assumes 25.8% bioavailability for dust; 18.3% bioavailability for soil
	mean	79.90	162.85		
	standard deviation	34.54	50.38		
	95% u c l of mean	97.81	188.97		
Area J	count	21	21	6.35E-05	assumes 25.8% bioavailability for dust; 18.3% bioavailability for soil
	mean	54.95	155.30		
	standard deviation	27.52	38.31		
	95% u c l of mean	65.31	169.72		
Area K	count	10	8	1.05E-04	assumes 25.8% bioavailability for dust; 18.3% bioavailability for soil
	mean	99.46	196.96		
	standard deviation	40.72	85.66		
	95% u c l of mean	123.06	254.36		
Total Pop	count	479	478	7.94E-05	assumes 25.8% bioavailability for dust; 18.3% bioavailability for soil
	mean	86.10	191.23		
	standard deviation	54.14	83.64		
	95% u c l of mean	90.17	197.52		

TABLE 5
CENTRAL TENDENCY EXPOSURE CALCULATIONS FOR RISKS IN COMMUNITY SOILS OU

		Dust Mean	Soil Mean	Risk	Bioavailability assumption
Area A	count	59	59	7.86E-06	assumes 25.8% bioavailable arsenic in dust; 18.3% bioavailable arsenic in soil
	mean	56.11	99.91		
	standard deviation	30.95	41.10		
	95% u c l of mean	62.85	108.86		
Area B	count	70	70	1.03E-05	assumes 25.8% bioavailable arsenic in dust; 18.3% bioavailable arsenic in soil
	mean	65.97	145.79		
	standard deviation	40.82	53.81		
	95% u c l of mean	74.12	156.53		
Area C	count	36	37	1.24E-05	assumes 25.8% bioavailable arsenic in dust; 18.3% bioavailable arsenic in soil
	mean	59.56	194.03		
	standard deviation	66.47	51.31		
	95% u c l of mean	78.29	208.28		
Area D	count	23	23	1.85E-05	assumes 25.8% bioavailable arsenic in dust; 18.3% bioavailable arsenic in soil
	mean	114.20	256.34		
	standard deviation	43.79	84.04		
	95% u c l of mean	129.88	286.43		
Area E	count	68	68	1.43E-05	assumes 25.8% bioavailable arsenic in dust; 18.3% bioavailable arsenic in soil
	mean	87.55	219.80		
	standard deviation	37.69	51.97		
	95% u c l of mean	95.18	230.32		
Area F	count	135	135	1.76E-05	assumes 25.8% bioavailable arsenic in dust; 18.3% bioavailable arsenic in soil
	mean	118.99	255.75		
	standard deviation	63.15	76.97		
	95% u c l of mean	127.93	266.64		
Area G	count	28	28	1.19E-05	assumes 25.8% bioavailable arsenic in dust; 18.3% bioavailable arsenic in soil
	mean	79.44	150.32		
	standard deviation	40.41	61.62		
	95% u c l of mean	92.44	170.16		
Area H	count	17	17	9.74E-06	assumes 25.8% bioavailable arsenic in dust; 18.3% bioavailable arsenic in soil
	mean	70.19	103.46		
	standard deviation	33.41	47.94		
	95% u c l of mean	84.33	123.77		
Area I	count	12	12	1.29E-05	assumes 25.8% bioavailable arsenic in dust; 18.3% bioavailable arsenic in soil
	mean	79.90	162.85		
	standard deviation	34.54	50.38		
	95% u c l of mean	97.81	188.97		
Area J	count	21	21	1.02E-05	assumes 25.8% bioavailable arsenic in dust; 18.3% bioavailable arsenic in soil
	mean	54.95	155.30		
	standard deviation	27.52	38.31		
	95% u c l of mean	65.31	169.72		
Area K	count	10	8	1.69E-05	assumes 25.8% bioavailable arsenic in dust; 18.3% bioavailable arsenic in soil
	mean	99.46	196.96		
	standard deviation	40.72	85.66		
	95% u c l of mean	123.06	254.36		
Total Pop	count	479	478	1.28E-05	assumes 25.8% bioavailable arsenic in dust; 18.3% bioavailable arsenic in soil
	mean	86.10	191.23		
	standard deviation	54.14	83.64		
	95% u c l of mean	90.17	197.52		

APPENDIX E
LEAD MODELING RUN

SUBAREA A

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 4.00 ug Pb/L DEFAULT
WATER Consumption: DEFAULT

SOIL & DUST:

Soil: constant conc.
Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	75.9	69.0
1-2	75.9	69.0
2-3	75.9	69.0
3-4	75.9	69.0
4-5	75.9	69.0
5-6	75.9	69.0
6-7	75.9	69.0

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 2.50 ug Pb/dL

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.6	4.76	1.74	2.62	0.38	0.00	0.02
1-2:	2.7	6.47	2.76	2.73	0.95	0.00	0.03
2-3:	2.6	6.91	2.78	3.08	0.99	0.00	0.06
3-4:	2.4	6.87	2.80				
4-5:	2.1	6.13	2.09				
5-6:	1.9	6.19	1.89				
6-7:	1.8	6.43	1.79				

SUBAREA A

3-4:	2.99	1.02	0.00	0.07
4-5:	2.91	1.06	0.00	0.07
5-6:	3.08	1.13	0.00	0.09
6-7:	3.40	1.15	0.00	0.09

SUBAREA B

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 4.00 ug Pb/L DEFAULT
WATER Consumption: DEFAULT

SOIL & DUST:

Soil: constant conc.
Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	256.6	146.8
1-2	256.6	146.8
2-3	256.6	146.8
3-4	256.6	146.8
4-5	256.6	146.8
5-6	256.6	146.8
6-7	256.6	146.8

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 2.50 ug Pb/dL

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)	
0.5-1:	4.1	7.52	4.59	
1-2:	4.5	10.80	7.23	
2-3:	4.2	11.31	7.31	
3-4:	4.0	11.35	7.40	
4-5:	3.4	9.55	5.59	
5-6:	2.9	9.30	5.07	
6-7:	2.7	9.38	4.80	
YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.54	0.37	0.00	0.02
1-2:	2.63	0.91	0.00	0.03
2-3:	2.98	0.96	0.00	0.06

SUBAREA B

3-4:	2.90	0.99	0.00	0.07
4-5:	2.85	1.04	0.00	0.07
5-6:	3.03	1.11	0.00	0.09
6-7:	3.36	1.13	0.00	0.09

SUBAREA C

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
 Indoor AIR Pb Conc: 30.0 percent of outdoor.
 Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 4.00 ug Pb/L DEFAULT
 WATER Consumption: DEFAULT

SOIL & DUST:

Soil: constant conc.
 Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	476.5	241.3
1-2	476.5	241.3
2-3	476.5	241.3
3-4	476.5	241.3
4-5	476.5	241.3
5-6	476.5	241.3
6-7	476.5	241.3

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

MATERNAL CONTRIBUTION: Infant Model
 Maternal Blood Conc: 2.50 ug Pb/dL

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	5.7	10.65	7.83	2.45	0.35	0.00	0.02
1-2:	6.4	15.65	12.23	2.51	0.87	0.00	0.03
2-3:	6.0	16.29	12.44	2.87	0.92	0.00	0.06
3-4:	5.7	16.48	12.65				
4-5:	4.8	13.55	9.67				
5-6:	4.1	12.96	8.80				
6-7:	3.7	12.87	8.36				

SUBAREA C

3-4:	2.81	0.95	0.00	0.07
4-5:	2.79	1.02	0.00	0.07
5-6:	2.98	1.09	0.00	0.09
6-7:	3.31	1.11	0.00	0.09

SUBAREA D

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
 Indoor AIR Pb Conc: 30.0 percent of outdoor.
 Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 4.00 ug Pb/L DEFAULT
 WATER Consumption: DEFAULT

SOIL & DUST:

Soil: constant conc.
 Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	419.4	216.7
1-2	419.4	216.7
2-3	419.4	216.7
3-4	419.4	216.7
4-5	419.4	216.7
5-6	419.4	216.7
6-7	419.4	216.7

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

MATERNAL CONTRIBUTION: Infant Model
 Maternal Blood Conc: 2.50 ug Pb/dL

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0-1:	5.3	9.86	7.01	2.47	0.36	0.00	0.02
1-2:	6.0	14.43	10.97	2.54	0.88	0.00	0.03
2-3:	5.6	15.03	11.14	2.90	0.93	0.00	0.06
3-4:	5.3	15.18	11.32				
4-5:	4.4	12.52	8.62				
5-6:	3.8	12.02	7.84				
6-7:	3.4	11.98	7.45				

SUBAREA D

3-4:	2.83	0.96	0.00	0.07
4-5:	2.81	1.03	0.00	0.07
5-6:	2.99	1.09	0.00	0.09
6-7:	3.32	1.12	0.00	0.09

SUBAREA E

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 4.00 ug Pb/L DEFAULT
WATER Consumption: DEFAULT

SOIL & DUST:

Soil: constant conc.
Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	581.7	286.5
1-2	581.7	286.5
2-3	581.7	286.5
3-4	581.7	286.5
4-5	581.7	286.5
5-6	581.7	286.5
6-7	581.7	286.5

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 2.50 ug Pb/dL

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	6.5	12.08	9.30
1-2:	7.3	17.84	14.49
2-3:	6.9	18.55	14.76
3-4:	6.5	18.82	15.05
4-5:	5.5	15.40	11.56
5-6:	4.6	14.67	10.54
6-7:	4.2	14.50	10.02

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.41	0.35	0.00	0.02
1-2:	2.47	0.85	0.00	0.03
2-3:	2.82	0.90	0.00	0.06

SUBAREA E

3-4:	2.76	0.94	0.00	0.07
4-5:	2.76	1.01	0.00	0.07
5-6:	2.95	1.08	0.00	0.09
6-7:	3.28	1.11	0.00	0.09

SUBAREA F1

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
 Indoor AIR Pb Conc: 30.0 percent of outdoor.
 Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 4.00 ug Pb/L DEFAULT
 WATER Consumption: DEFAULT

SOIL & DUST:

Soil: constant conc.
 Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	534.0	266.1
1-2	534.0	266.1
2-3	534.0	266.1
3-4	534.0	266.1
4-5	534.0	266.1
5-6	534.0	266.1
6-7	534.0	266.1

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

MATERNAL CONTRIBUTION: Infant Model
 Maternal Blood Conc: 2.50 ug Pb/dL

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	6.1	11.44	8.64	2.42	0.35	0.00	0.02
1-2:	6.9	16.86	13.48	2.49	0.86	0.00	0.03
2-3:	6.5	17.54	13.72	2.84	0.91	0.00	0.06
3-4:	6.2	17.77	13.97				
4-5:	5.2	14.56	10.71				
5-6:	4.4	13.90	9.76				
6-7:	3.9	13.77	9.27				

SUBAREA F1

3-4:	2.78	0.95	0.00	0.07
4-5:	2.77	1.02	0.00	0.07
5-6:	2.96	1.08	0.00	0.09
6-7:	3.29	1.11	0.00	0.09

SUBAREA F2

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
Indoor AIR Pb Conc: 30.0 percent of outdoor.

Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 4.00 ug Pb/L DEFAULT
WATER Consumption: DEFAULT

SOIL & DUST:

Soil: constant conc.

Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	508.1	254.9
1-2	508.1	254.9
2-3	508.1	254.9
3-4	508.1	254.9
4-5	508.1	254.9
5-6	508.1	254.9
6-7	508.1	254.9

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 2.50 ug Pb/dL

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	5.9	11.09	8.28	2.43	0.35	0.00	0.02
1-2:	6.7	16.32	12.92	2.50	0.86	0.00	0.03
2-3:	6.3	16.98	13.15	2.86	0.92	0.00	0.06
3-4:	6.0	17.19	13.38				
4-5:	5.0	14.11	10.24				
5-6:	4.3	13.48	9.33				
6-7:	3.8	13.37	8.86				

SUBAREA F2

3-4:	2.79	0.95	0.00	0.07
4-5:	2.78	1.02	0.00	0.07
5-6:	2.97	1.09	0.00	0.09
6-7:	3.30	1.11	0.00	0.09

SUBAREA I.

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
 Indoor AIR Pb Conc: 30.0 percent of outdoor.
 Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 4.00 ug Pb/L DEFAULT
 WATER Consumption: DEFAULT

SOIL & DUST:

Soil: constant conc.
 Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	75.0	68.7
1-2	75.0	68.7
2-3	75.0	68.7
3-4	75.0	68.7
4-5	75.0	68.7
5-6	75.0	68.7
6-7	75.0	68.7

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

INTERNAL CONTRIBUTION: Infant Model
 Maternal Blood Conc: 2.50 ug Pb/dL

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.6	4.75	1.73	2.62	0.38	0.00	0.02
1-2:	2.7	6.45	2.74	2.73	0.95	0.00	0.03
2-3:	2.6	6.89	2.75	3.08	0.99	0.00	0.06
3-4:	2.4	6.85	2.78				
4-5:	2.1	6.12	2.08				
5-6:	1.9	6.17	1.88				
6-7:	1.8	6.42	1.77				

SUBAREA I

3-4:	2.99	1.02	0.00	0.07
4-5:	2.91	1.06	0.00	0.07
5-6:	3.08	1.13	0.00	0.09
6-7:	3.40	1.15	0.00	0.09

SUBAREA J

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
 Indoor AIR Pb Conc: 30.0 percent of outdoor.
 Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 4.00 ug Pb/L DEFAULT
 WATER Consumption: DEFAULT

SOIL & DUST:

Soil: constant conc.
 Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	191.2	118.6
1-2	191.2	118.6
2-3	191.2	118.6
3-4	191.2	118.6
4-5	191.2	118.6
5-6	191.2	118.6
6-7	191.2	118.6

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

MATERNAL CONTRIBUTION: Infant Model
 Maternal Blood Conc: 2.50 ug Pb/dL

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	3.6	6.54	3.58
1-2:	3.9	9.27	5.65
2-3:	3.6	9.75	5.70
3-4:	3.4	9.76	5.76
4-5:	2.9	8.33	4.34
5-6:	2.6	8.18	3.93
6-7:	2.4	8.32	3.72

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.57	0.37	0.00	0.02
1-2:	2.66	0.92	0.00	0.03
2-3:	3.02	0.97	0.00	0.06

SUBAREA J

3-4:	2.93	1.00	0.00	0.07
4-5:	2.87	1.05	0.00	0.07
5-6:	3.05	1.12	0.00	0.09
6-7:	3.37	1.14	0.00	0.09

OPPORTUNITY

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 4.00 ug Pb/L DEFAULT
WATER Consumption: DEFAULT

SOIL & DUST:

Soil: constant conc.
Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	134.0	94.0
1-2	134.0	94.0
2-3	134.0	94.0
3-4	134.0	94.0
4-5	134.0	94.0
5-6	134.0	94.0
6-7	134.0	94.0

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 2.50 ug Pb/dL

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	3.1	5.67	2.68	2.59	0.38	0.00	0.02
1-2:	3.3	7.90	4.23	2.70	0.93	0.00	0.03
2-3:	3.1	8.35	4.26	3.05	0.98	0.00	0.06
3-4:	2.9	8.34	4.30				
4-5:	2.5	7.24	3.23				
5-6:	2.3	7.20	2.92				
6-7:	2.1	7.39	2.76				

OPPORTUNITY

3-4:	2.96	1.01	0.00	0.07
4-5:	2.89	1.06	0.00	0.07
5-6:	3.06	1.12	0.00	0.09
6-7:	3.39	1.14	0.00	0.09

ALL AREAS

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 4.00 ug Pb/L DEFAULT
WATER Consumption: DEFAULT

OIL & DUST:

Soil: constant conc.
Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	364.0	192.9
1-2	364.0	192.9
2-3	364.0	192.9
3-4	364.0	192.9
4-5	364.0	192.9
5-6	364.0	192.9
6-7	364.0	192.9

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

INTERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 2.50 ug Pb/dL

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0-1:	4.9	9.08	6.20	2.49	0.36	0.00	0.02
1-2:	5.5	13.22	9.73	2.57	0.89	0.00	0.03
2-3:	5.1	13.79	9.86	2.93	0.94	0.00	0.06
3-4:	4.9	13.90	10.00				
4-5:	4.1	11.52	7.60				
5-6:	3.5	11.10	6.91				
6-7:	3.2	11.10	6.55				

ALL AREAS

3-4:	2.86	0.97	0.00	0.07
4-5:	2.82	1.03	0.00	0.07
5-6:	3.00	1.10	0.00	0.09
6-7:	3.33	1.12	0.00	0.09